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# THE VETERINARY JOURNAL

## A Monthly Review of Veterinary Science.

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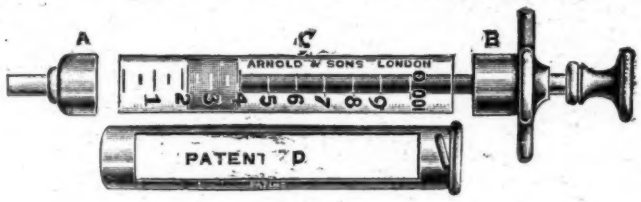


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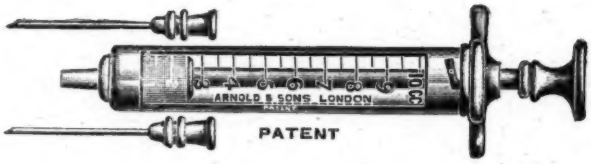


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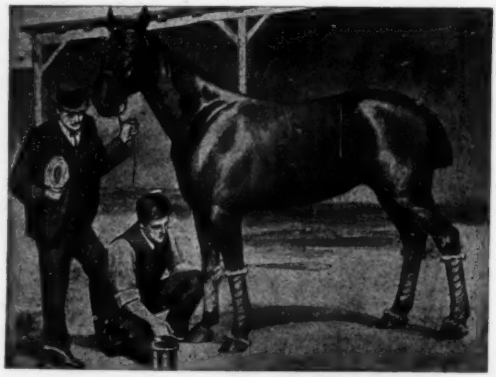

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# VETERINARY JOURNAL

OCTOBER, 1910.

## Editorials.

### THE EXPORTATION OF HORSES.

THE Exportation of Horses Order of 1910 (No. 2), which comes into force this month, is a step in the right direction, and should do much to prevent unnecessary suffering on the part of horses being shipped from Great Britain to the Continent of Europe. By this order all horses, asses, and mules, with certain exceptions, will have to be examined by a veterinary inspector and certified fit to be shipped. For each animal examined a fee of two shillings and sixpence must be paid before the examination takes place. The exemptions consist of valuable animals intended for breeding, racing, or exhibition, and also animals being sent to any port that is not in Europe, but in all these cases a permit from the Board will be necessary. In fact no horse, ass, or mule can be shipped without either a permit or a certificate from the veterinary inspector. The order has to be enforced by the Local Authority of the district in which the port of embarkation is situate.

This order is one which appears likely to have nothing but good results. It is not likely to interfere in any serious way with legitimate horse traffic, and it will undoubtedly put a stop to the transportation of horses in an unfit state to travel. It will also put an end to those most irritating prosecutions in which the voice of a lay inspector of the R.S.P.C.A. is so often raised in conflict with that of a competent veterinary surgeon, for while both

parties are desirous of preventing cruelty, opinions differ as to whether cruelty exist in the particular cases. The worst part of such cases is that one so often meets with magistrates who prefer to believe the word of an uneducated lay inspector to that of a highly trained veterinary expert. It seems difficult to persuade some people that a horse may be mechanically lame, and be able to stand or walk about perfectly free from any pain. In such cases it is absurd to say that cruelty is inflicted if a horse is walked about or made to stand on board ship. We all know men who are lame and who go about their business daily without any pain or other inconvenience than the mechanical interference with gait. It is strange that so many people are apparently unable to transfer the same line of reasoning to the horse, and especially so when they are assured by experts that so many cases are quite painless.

We feel sure that the new Order will do all that is necessary for the protection of horses about to be exported, and at the same time safeguard those dealers legitimately engaged in the business from unwarranted persecution by lay inspectors. On the other hand, nobody will be more glad than veterinary surgeons that a stop will now be definitely put to that gross cruelty, which not infrequently occurred hitherto by unscrupulous low-class dealers engaged in the decrepit horse traffic.—G. H. W.

---

## THE RELATION OF UDDER DISEASES OF THE COW TO SCARLET FEVER, DIPHTHERIA, AND SORE THROATS OF THE HUMAN SUBJECT.

THE very able paper on the above subject by Professor Gofton, which we reproduce in this issue of the VETERINARY JOURNAL, makes very interesting reading. It reopens an old discussion on subject-matter which is by no means settled in the minds of many veterinary surgeons and medical men. Professor Gofton has really no new evidence to bring forward, but his paper consists of a very able summary of views expressed by various authorities, and on which his conclusions are based.

There is very little room to doubt that each of the diseases

referred to may be disseminated by milk, but we agree with Professor Gofton that that is a very long way from proving that they are diseases of the cow transmitted to human beings.

It has frequently been asserted that contagious mammitis is one of the causes of sore throat in the human subject, because a somewhat similar streptococcus can be demonstrated in each case. Direct evidence of actual transmission, or satisfactory experimental proof, are wanting also. We, ourselves, were included in a number of persons who unwittingly put the matter to something in the nature of a test. An apparently healthy milch cow was brought in to a certain institution, and her milk was freely partaken of for several days by six persons—two adults and four children. Then two others took some of the milk and noticed a somewhat extra salty taste, as the result of which an examination was made, and the cow was found to be suffering from mammitis. The milk was shown to be highly charged with streptococci. In none of the eight persons who had partaken of the milk was there the slightest ill-effect observed, either in the way of sore throat or otherwise, although six persons had been drinking that milk for several days. We fully realize that this little involuntary experiment had negative results and that it actually *proves* nothing. But it must be regarded as fairly strong evidence to support the contention that sore throat cannot be commonly caused by drinking milk from cows affected with streptococcus mammitis. With regard to Dr. Klein's experiments and his conclusions we agree with Professor Gofton, and regard them as very far from conclusive.—G. H. W.

## General Articles.

# THE RELATIONSHIP BETWEEN SCARLET FEVER, DIPHTHERIA, AND SORE THROAT OF MAN, AND DISEASES OF THE UDDER AND TEATS OF COWS.

By A. GOFTON, M.R.C.V.S.

*Professor in the Royal Dick Veterinary College, Edinburgh.*

The question of a causal relationship between disease of the cow and scarlet fever of man formed the subject of a heated controversy in 1885 and subsequent years, but for a considerable period of time it has been in a condition of at least suspended animation. Interest in the matter, however, has been in some measure reawakened by the publication last year of a report by Drs. Hamer and Jones with reference to an extensive milk-borne outbreak of scarlet fever in London and Surrey, which they concluded was attributable to an eruptive disorder of the teats and udders of the cows producing the incriminated milk.

If the cow be really the natural subject of a disease capable of causing scarlet fever in man, the necessity for its immediate recognition in the interests of public health cannot be overestimated, but it is well known that ever since the bovine scarlatina theory was first promulgated, veterinarians have been unwavering in their opposition to it. It is the purpose of this paper to discuss the relationship which exists between scarlet fever, diphtheria, and sore throat of man, and diseases of the udder and teats of cows, and to see how far the theory of the animal origin of these diseases has been supported by facts brought to light by the many inquiries which have made during the period of some thirty years that has elapsed since it was first advanced.

It is of interest to refer for a few moments to the history of milk-borne epidemics, particularly with reference to the possibility of animal infection. It is undisputed, and has long been recognized that certain diseases of the cow are communicable to man, but the part which milk may play as a medium in the spread of contagious diseases other than those from which the cow is known to suffer does not appear to have received much attention until 1857. In that year Dr. Taylor, of Penrith, published what in all probability is the first record of a milk-borne epidemic, the disease being typhoid fever. In the light of our present knowledge the part played by the milk in this instance leaves practically no room for doubt, but at the time Dr.

Taylor's conclusions were questioned. His view, however, that milk could be infected by contact with persons affected with certain contagious diseases, slowly, but certainly, became an established fact, the last remnants of doubt being effectually dispelled by the introduction, at a later date, of compulsory notification with its resulting more regular and systematic inquiry into the origin of epidemics and the means by which they were spread.

It is not in the least surprising that in the course of a large number of investigations of milk-borne epidemics, the most searching inquiries have failed at times to trace the original source of infection, although a definite relationship may have been comparatively easily established between the epidemic and the milk supply. It was such failure which originally gave rise to the suggestion that the cow might be responsible for the infectivity of the milk when infection was traceable back to the farm on which it had been produced, but no human agency to which it could be attributed was discoverable. Thus it was that Power, in investigating the 1878 epidemic of diphtheria at Kilburn and St. John's Wood, submitted the hypothesis of a cow origin, which appears to have been based on an inability to demonstrate the actual source of infection, and not upon the co-existence of any specific disease amongst the cows. Similarly, with the North London epidemic of scarlet fever in 1882, the same official writes[1]: "In the end I found myself as on a former occasion, called upon to face the question whether or not actual cow conditions might have been competent for the results observed, and in considering the question I came to see that a hypothesis of cow causation would fit the facts that needed explanation as well as, or even better than, any other hypothesis." He found that the only new element that had been introduced into the milk business for a considerable period before the milk became infective was a cow which had been some ten days overdue in calving, and whose milk had come into ordinary use three or four days later, and almost coincidently with the commencement of the outbreak, though there is a decided vagueness as to the actual date on which the milk was first used. Some five weeks after the commencement of the epidemic, Power examined this cow, and he noted that "she had here and there lost portions of her coat, and that her buttocks and posterior udder were fouled with excremental matter and perhaps vaginal discharge as well." He proceeds: "That if scarlatina have other animal source

[1] Report to the Local Government Board. Power.

than human source, it may be that one such source is the cow that has recently calved, a cow either not at all ill (except for her parturition), or not so obviously ill as would prevent her milk being used for human consumption." These appear to be the facts on which his hypothesis of the bovine origin of the outbreak were based, and on which the proposition was first seriously advanced that scarlet fever of man might have an animal origin.

This case had an important result. It led to a series of experiments being carried out by Klein, on behalf of the Local Government Board, with the view of determining the susceptibility of the cow to infection with human scarlatina, and to the issuing of instructions to the inspectors of the Board for the making of careful investigations and observations in order to discover if confirmation of Power's hypothesis was obtainable. No confirmation was forthcoming until Power discovered the disease amongst the cows at Hendon, which he, in conjunction with Klein, concluded was scarlatina of the cow, but which, on the other hand, the Agricultural Department of the Privy Council as a result of subsequent investigations just as emphatically declared bore no etiological relationship to the human scarlatina. The Hendon case was followed by a series of milk-borne epidemics which were attributed to more or less ill-defined diseases of the cow, and, similarly, diphtheria and sore throat epidemics came to be regarded in certain circles as having not infrequently a similar origin. As already stated, it is my object to see how far the facts since brought to light lend support to the theory of the animal origin of these diseases.

It is my intention to take up the three conditions separately, dealing first with scarlet fever.

### Scarlet Fever.

The cow disease at Hendon is always quoted as the typical example of the alleged bovine scarlatina, and I purpose giving a description of it in order to see how far it supports the view of the occurrence of a specific disease in cows differing from other previously recognized bovine diseases. According to Klein, it presented the following clinical features: It was essentially contagious and easily conveyed from one cow to another, especially by the hands of the milkers. It was characterized by a vesico-pustular eruption on the udders and teats, commencing with red raised papules, and much injection of the skin. The pustules became covered with brown crusts, which if removed exposed a bleeding sore, but if left untouched

thickened and ultimately became loose, falling off in from ten to fifteen days, and leaving a contracting healing sore. A slightly raised reddish scar marked the site of the lesion when completely healed. Ulceration, scabs, scurfiness, and loss of hair in patches on different parts of the skin accompanied the udder lesions. The cows had a slight cough and lost condition, but there was no fever and no apparent general disturbance of health.

None of these symptoms individually, nor the whole of them collectively, enable one to diagnose the condition. The description of the udder lesion is equally applicable to more than one of the well-known eruptive conditions met with in milk cows, and at the time of the "Hendon disease" members of the veterinary profession were unable to discover any character by which it could be distinguished from similar eruptive conditions prevailing at the same time in other cowsheds, and which were not associated with scarlet fever. Klein, however, held that the "Hendon disease" possessed differentiating features in the duration of the eruption, the regularity of contour of the lesions, the thickness of the scabs, the amount of induration round the sores, and the extent to which they were raised above the surrounding skin. These differences are, however, merely variations in degree, and are too ill-defined to give them a diagnostic value. In any extensive outbreak of cow-pox, variations equal in degree to those claimed as differentiating features may be found, depending on the severity of the attack, whether the vesicles of the sore remain discrete or become confluent, the amount of irritation caused by milking, and possibly also to secondary infection of the sores as the result of rupture of the vesicles or the removal of the scabs in the act of milking.

From the point of view of diagnosis, Klein at various times laid much emphasis on the co-existence of ulceration, scabs, and scurfiness of the skin with the loss of hair in patches. I do not propose discussing the various causes which may give rise to such lesions of the skin—suffice it is to say that the veterinarian was not then and is not now conversant with any disease of the cow characterized by the co-existence of these udder and skin lesions. Indeed, some doubt must remain as to whether skin lesions were even a constant feature of the cow disease at Hendon, for Professor Axe in his report to the Agricultural Department of the Privy Council of his investigations into the nature of the cow disease says: "In some exceptional cases it is stated to have been associated with scaliness and thickening of the skin about the quarters, and with loss of hair in patches." Whether

skin lesions were a constant feature of the Hendon disease or not, medical and lay inspectors in the course of subsequent investigations certainly arrived at a confident diagnosis of cow scarlatina in which no skin lesions were present, and in which neither cough nor loss of condition of the animals held responsible for the infectivity of the milk is recorded. Thus, in the report of the 1909 London epidemic it is stated that[1] "in no instance did we discover evidence of loss of hair in patches," and in the Glasgow report (1892)[2] "skin lesions were also absent."

Notwithstanding his assertions of the existence of differentiating features between the "Hendon disease" and other eruptive conditions of the teats and udders of cows Klein himself appears to have been unable to distinguish between them from their clinical appearances. In the Glasgow epidemic (1892) it was only after a series of inoculation tests that Klein discovered that the cows were affected with cow-pox, though because he found what he regarded as his specific streptococcus alongside several other species of organisms in lymph from the cows' teats, and produced a pustular eruption by the inoculation of calves with this contaminated lymph, the conclusion appears to have been arrived at that the cows were suffering from a mixed infection.

In some instances neither udder nor skin lesions appear to have been regarded as necessary to justify a diagnosis of bovine scarlatina. Dr. Parsons, in his report of the Macclesfield epidemic (1889) states that[3]: "The only circumstance that I have been able to discover fulfilling in point of time the requirements of the case is the addition on January 23 to the general stock of milk of that yielded by the cow which calved on January 20, and the only explanation which suggests itself as to how the milk of this particular cow came to have infectious properties is that the cow herself may have developed in the puerperal condition some ailment, so trivial, so evanescent as to have escaped the notice not only of her owner but also of the eminent veterinary authorities who at a later stage examined her." It is perhaps advisable to mention that the medical inspectors also examined the cows and were equally unable to discover any signs of illness amongst them. A postscript to the report states that the cows in question developed some pimples and vesicles on one of her teats some six weeks after the commencement of the human epidemic. She was

[1] Report of Drs. Hamer and Jones, 1909.
[2] Report of Drs. Russell and Chalmers.
[3] Report to the Local Government Board.

then suckling a calf which remained healthy, and the eruption did not spread to the other cows on the premises. It was therefore essentially different from the "Hendon disease." Notwithstanding, this case appears to have been confidently regarded by the Local Government Board officials of the time as one in which the responsibility for the human epidemic was brought home to the cow.

It must be obvious from what has been said that the alleged bovine scarlatina does not possess any characteristics which would permit of its diagnosis by any ordinary clinical examination.

The *post-mortem* appearances, as described by Klein, are of no greater value in establishing the occurrence of a specific disease. The Hendon cows which were examined showed on *post mortem* the following appearances: Slight pleuritis with recent fibrinous deposits and adhesions, congestion and extravasation in some of the lobules of the lungs, discoloured and softened patches in the liver, ecchymoses and petechiæ in the substance and in the capsule of the spleen, swelling and extravasation of blood in the lymphatic glands, congestion and glomerulo-nephritis of the cortex of the kidneys. However close the resemblance between these *post-mortem* appearances and those of scarlet fever of man, they are of little value as evidence of identity of the animal of human diseases, for exactly similar conditions are to be found in many septic diseases which do not bear the slightest resemblance to the alleged bovine scarlatina or to scarlatina of man.

The bacteriological investigations on which Klein based his theory of bovine scarlatina are equally unconvincing. Klein isolated a streptococcus from human scarlatina which he regarded as its essential causal agent. From the Hendon cows he also isolated a streptococcus from human scarlatina which he regarded as its essential causal agent. From the Hendon cows he also isolated a streptococcus which he concluded was responsible for the cow disease, and which he claimed to be identical with the streptococcus isolated from human scarlatina. He also claimed that by the subcutaneous inoculation of either of these streptococci the disease was reproduced in the cow and the bacteria were recovered from the teat and udder lesions of the experimental animals. He therefore concluded that the cow disease was caused by the same organism as human scarlatina, and that, in fact, it was scarlatina of the cow. If Klein's conclusions were correct it is most extraordinary that in the twenty-five years which have elapsed since he carried out his experiments no confirmation of his results has been forthcoming. To accept his conclusions as correct also means acceptance of the view that with the discovery of his streptococcus he

demonstrated the specific causal agent of scarlet fever. It is difficult to understand on what ground Klein thought himself justified in forming the opinion that his streptococcus was the causal agent of scarlet fever. Up to the present day, notwithstanding the advantage of a vastly greater knowledge of bacteriology, it has not been found possible to connect streptococci etiologically with scarlat fever. They are found with great constancy in the fauces in that disease and are responsible for most of its complications, but streptococci, indistinguishable from them, are also met with in the throat in other acute catarrhal conditions, and it seems highly probable that their effects in scarlet fever are secondary only to the action of a specific virus whose nature has yet to be determined.

It is remarkable that Klein's experiments to establish a relationship between the cow disease at Hendon and human scarlatina were limited to the injection of streptococci isolated from the cows, and those which he assumed to be the cause of scarlet fever. Had his results been tested by the experimental treatment of cows with scarlatinal material which undoubtedly possessed infective properties, and been found to coincide with those following the inoculation of the streptococci, his conclusions would have been placed on a firm basis. But we are not without knowledge of the results of the experimental treatment of cows with scarlatinal material. Klein, himself, in 1882, on behalf of the Local Government Board, attempted to infect cows with scarlet fever by inoculating and feeding them with scarlatinal material. His experiments absolutely failed, the only result being the production of a localized abscess following the subcutaneous injection of muco-purulent throat discharge—a not surprising result. Later, McFadyean carried out similar experiments on behalf of the Agricultural Department of the Privy Council, his experiments including the introduction of a large quantity of infective material into the genital passages of a newly calved cow. His attempts to infect the cow also proved abortive. A number of experiments were carried out by a committee on behalf of the Edinburgh Medico-Chirurgical Society with a similar object and a similar result. Loeffler, in Germany, also failed to produce the disease in cows by feeding and inoculation experiments. Moreover, not a single instance has been produced of the development of Hendon or other disease of the cow, as the result of the contact of attendants affected with scarlet fever, or who have been in close contact with scarlatina patients and were known carriers of infection. This is not because of the lack of occasion, for opportunities of infection in this way have occurred time and again.

The appearance of the cow disease, if it be really scarlatinal in nature, must bear a distinct relationship so far as time is concerned with the appearance of the resulting human disease, but a time relationship, though a point of the highest importance in establishing an etiological connection between the two, has never been shown to exist. Even in the North London case attributed to the cow disease at Hendon, and on which the foundations of the bovine scarlatina theory may be said to have been laid, it is clear from the recorded dates, which were undisputed, that the cow disease was in existence on the Hendon farm for a fortnight before the commencement of the human disease, the incubative period of which varies from one to six days. The Glasgow epidemic of 1892 is even more striking, the cow disease to which the infectivity of the milk was attributed having been in existence for a month before the the milk became infective. Further comment is unnecessary, it must rest with those who support the theory of cow scarlatina to explain the non-infectivity of the milk between the appearance of the cow disease and the commencement of the human epidemic.

Before leaving this point it is necessary to refer to the London epidemic of 1909, because the reporters assert that the appearance of the cow disease and the human epidemic synchronized. One searches the report in vain, however, for evidence in support of this conclusion, which is based on the assumption that the disease was introduced into the herd by a particular newly calved heifer. There is not a single scrap of evidence to show that she introduced the disease, and that she was not infected after her introduction. There is room for the greatest doubt as to the correctness of the author's conclusions both on this point and as to the date on which the milk of this heifer is said to have been first used. For five or six weeks after the opening of the inquiry, the milkers contradicted one another as to the date on which her milk was first added to the general stock, and the only ascertained facts with reference to this heifer were that she had calved about May 24, and that her calf had died four or five days later, which means, having regard to the customs prevailing on the farm, that her milk would first be used a week before the date (June 7) fixed by the authors, and the same length of time before the milk was regarded as having become infective.

Perhaps the weakest point in the whole of the case against the cow lies in the fact that in not a single instance has any explanation, which will bear investigation, been offered of the manner in which the cows contracted the disease. Spontaneity of origin must be negatived.

In the absence of the introduction of new stock, a suggestion of human infection must also be negatived, for in every instance in which scarlet fever has been attributed to animal infection, human sources of infection have been first sought for, and it is believed eliminated. In more than one case the alleged bovine scarlatina has been introduced by a newly purchased cow, and in not one single instance has a scarlatinal epidemic existed in the district from which the cow has been removed, nor amongst the consumers of the milk of her former companions similarly affected. The Hendon case again affords an excellent illustration. The cow disease alleged to have caused the human scarlatina was admittedly introduced into the Hendon cowshed by three cows purchased from a lot of thirty belonging to a dealer. The remaining twenty-seven cows, the majority of which were affected with an udder condition indistinguishable from that of the Hendon cows, were sold in lots of from one to eight within a few days, and distributed over a number of cowsheds. Both before and after sale and throughout the whole period of the cow disease, with which they infected their new companions the milk from all the cows was sold in the ordinary way. Notwithstanding the most careful inquiry in the districts to which their milk was consigned, no complaint had arisen as to its wholesomeness, no case of scarlet fever attributable to milk could be found, and the medical officers of health of the districts certified to the absence of any epidemic at the time.[1] Again, if cows are affected with a disease capable of giving rise to scarlet fever in man, the sale and distribution of the affected animals should be followed by a series of scarlatinal epidemics in the new districts in which their milk is distributed. But this has not been so. The Wimbledon and Merton epidemic of 1886, which was attributed to disease of the cows, may be cited in illustration. On visiting the farm involved, Power found that the whole herd of forty-one cows had been sold, and seventeen had already been removed from the premises, and[2] "of those remaining, some few appeared to be recovering from an affection of the skin and udder, very similar to the malady reported on by Dr. Klein as having occurred amongst certain cows at Hendon." In view of the fact that scarlet fever did not follow the distribution of these cows, it must have required a considerable amount of courage on the part of the inspector to attribute the infectivity of the milk to disease of the cows.

In the report of the London epidemic (1909) the suggestion is

[1] Report of Professor Axe to Agricultural Department of Privy Council.
[2] Report to the Local Government Board.

made that the bovine scarlatina had its origin in the use of feeding cakes, a suggestion which is altogether unworthy of its authors, and one which is so improbable that it does not merit discussion.

The position for and against the theory of bovine scarlatina may be summarized as follows :—

*For.*—Failure in certain milk-borne epidemics of scarlet fever to trace human infection.

The co-existence of scarlet fever in man, and of an eruptive disorder of the teats and udders of cows.

Klein's discovery of a streptococcus in the lesions of the teats and udders of certain cows at Hendon which he regarded as identical with a streptococcus isolated from human scarlatina and assumed to be the cause of that disease. With both streptococci he claimed to be able to reproduce the cow disease.

The similarity of the *post-mortem* appearances of the Hendon cows and of Klein's experimental animals with those of human scarlatinal patients.

*Against.*—It requires more than the failure to trace infection to human sources to justify, attributing it to animal sources.

The human and animal diseases have not occurred synchronously.

There is no reason for believing Klein's streptococcus to be the specific organism of scarlet fever.

The *post-mortem* appearances are common to a number of dissimilar diseases.

All attempts to infect cows experimentally with scarlet fever have failed, and there is no known case of infection of cows as the result of accidental contact with infected persons.

Veterinarians, British and Continental, are unanimously of opinion that the cow is not the subject of scarlatina.

In conclusion, the following opinions of British and Continental authorities may be quoted:—

"The alleged cow scarlatina has failed to establish a place for itself in veterinary pathology, and except among medical officers of health in Great Britain, belief in the occurrence of such a source of human outbreaks has within recent years steadily lost ground." —McFadyean, *Journ. Comp. Path. and Therap.*, vol. xxii., p. 341.

"After an examination of the evidence on both sides of the question, we are of opinion that the case for cow scarlatina was not proved."—Swithinbank and Newman, "Bacteriology of Milk," p. 289.

"Scarlet fever was not communicable from man to animals. . . .

With regard to Klein's experiments, the general opinion in Germany was that Dr. Klein had confounded the disease in question (Hendon disease) with either cow-pox or foot-and-mouth disease."—Ostertag, Seventh International Congress on Hygiene, 1891."

"It is strange that this old fallacy should have recently again cropped up by the assertion that scarlet fever of man is produced by the milk of cows suffering from scarlet fever. . . . We may point out that cows are absolutely immune to scarlet fever."—Friedberger and Fröhner, "Veterinary Pathology" (Haye's transl.), vol. ii., p. 650.

"About eighteen years ago some scarlet fever epidemics aroused great attention in England . . . from the evidence that is now available, one is justified in regarding it as established that the outbreaks of scarlet fever in question had no relation to disease in cows."—Jenson, "Milk Hygiene" (Pearson's transl.), p. 115.

### Diphtheria.

If it can be said with truth of scarlet fever that belief in the bovine origin of such human outbreaks has been steadily declining during recent years, the remark may be made with still greater truth of diphtheria. The fact that the causal agent of this disease is well known and recognizable, has contributed in no small degree to this change of opinion. In part also it is due to the fact, unknown and probably unthought of, in the early days of the bovine diphtheria hypothesis, that patients recovered from diphtheria very commonly harbour the causal organism of that disease in the throat or nose for several weeks, sometimes for indefinite periods varying from one hundred to three hundred days, and that so long as the organisms remain present, the patient, though apparently completely recovered, continues to act as a centre of infection. These organisms are frequently of low virulence, but under favourable conditions are capable of returning to the more virulent type.

Further, it is now known that the same organism may be found in the throat or nose of those who have been nursing diphtheria patients, or in close contact with them, and also, though with much less frequency, in the throats of apparently healthy persons who have not suffered from diphtheria, and who, so far as is known, have not been exposed to infection.

A series of diphtheritic, though apparently innocent sore throats, whose real nature can only be determined by a bacteriological examination, are found in association with all diphtheria epidemics,

and the hosts of the bacillus are sometimes inconvenienced by its presence to such a small degree that their daily duties are performed without interruption, and they are regarded merely as the victims of a slight catarrhal cold.

These facts are placed before you because the bovine diphtheria hypothesis had its inception originally in a failure to trace infection of milk to human agencies, and they illustrate the extreme difficulty which exists in completely eliminating human sources of infection. They explain the ease with which the diphtheria bacilli may be transferred from persons apparently healthy, or, as more frequently happens, affected with what appears to be a slight harmless cold, to lesions or sores on the teats of cows in which their presence may be demonstrated, but for the occurrence of which they are in no way responsible. They emphasize the necessity of experimental tests in all cases in which diphtheria bacilli are found in abnormal teat or udder conditions, to show whether their presence is accidental or the essential cause of the cow condition. Without experimental tests there is absolutely no justification for the conclusion that the abnormal conditions of the udders and teats of cows are the result of the action of diphtheria bacilli because they have been found in the lesions. It is my opinion that experimental tests will, as they have done in the past, absolutely fail to produce evidence in support of the view that cows are naturally the subject of a disease caused by the Klebs-Loeffler bacillus, and capable of infecting human beings with diphtheria through the agency of the milk.

In the earliest days of the bovine diphtheria hypothesis, mammitis was regarded as capable of inducing diphtheria amongst the consumers of the milk with which the abnormal secretions of the diseased gland had been mixed. The epidemic at Rugby school in 1881 was so attributed, but at that time bacteriology was in its infancy, and the Klebs-Loeffler bacillus as the cause of diphtheria unknown. Since that time many investigations have been made into the cause of mastitis in cows, without having produced any evidence to show that the diphtheria bacillus is ever a cause of mastitis, or that any etiological relationship has ever at any time existed between inflammation of the cow's udder and diphtheria of man. Exceptionally, diphtheroid bacilli, morphologically indistinguishable from the diphtheria bacillus, have been found in association with other organisms in the secretions from, and in the parenchyma of, inflamed udders of dairy cows, but so far as the question at present under consideration is concerned, their presence has no

significance, because cultural and inoculation tests have shown them to be essentially different from the causal bacillus of diphtheria. More frequently similar diphtheroid bacilli, differing from the diphtheria bacilli in their cultural characters, and in their pathogenicity for animals, have been isolated from sores on the teats and from healthy teats of apparently perfectly healthy cows.

Klein, in the reports to the Local Government Board on his investigations into the cause of diphtheria (1888-89, 1889-90), was the first to bring forward what appeared to be material evidence in support of the hypothesis that the cow was the subject of diphtheria, and by means of her milk able to convey the disease to man. He held that diphtheria of the cow was manifested by an eruption on the teats and udders of the animals affected, and claimed to have isolated the bacillus from the teat eruptions and from milk obtained direct from the gland uncontaminated from outside sources. He based this view on the results of the experimental inoculation of cows with bacilli isolated from human diphtheritic membranes. The inoculations were made into the subcutaneous tissue of the shoulder, and resulted in a painful diffused swelling at the seat of the inoculation and beginning about the fifth day, in the appearance of a vesico-pustular eruption on the skin of the udder and teats, running its course in from six to eight days, and appearing in successive crops. The experimental cows were obviously ill, they showed an evanescent rise of temperature on the second or third day, and the majority either died or were killed in a more or less moribund condition after ten to twenty-five days. The bacilli were recovered from the udder lesions, and in two cases from the milk. The udder eruption was not constant; it appeared in four out of eight cows inoculated.

These experiments of Klein's cannot be regarded as of any value for or against the bovine diphtheria theory for several reasons. In the 1888 portion of his report Klein casts doubt on the specificity of the Klebs-Loeffler bacillus, and the organism with which he worked varied in its cultural characters from those of the bacillus now universally recognized as the organism of diphtheria, notably in growing well on gelatine at 20° C. A feature of the Klebs-Loeffler bacillus appears to be its localization to the seat of inoculation, whilst a striking result of Klein's experiments was that the bacilli entered the circulation and appeared in the udder eruption, and in two cases in the milk. The limited number of his experiments on cows and his inconstant results scarcely justified the general conclusion that a vesico-pustular eruption was a feature of bovine diphtheria, assuming that such a disease

exists. Subsequent investigators have failed to confirm Klein's results. It is therefore not surprising that his conclusions have come almost universally to be regarded as having been founded on error.

It was nevertheless characteristic of the period in which Klein carried out his investigations to attribute the origin of milk-borne diphtheria as well as other milk-borne epidemics to co-existent eruptive disorders of the udders and teats of dairy cows; and in 1890 Klein refers to two such outbreaks at Croydon and Bishop's Stortford, in which the cows on the farms supplying the infected milk were affected with [1] "a contagious, ulcerative eruption on the teats very like that occurring in the cows experimentally inoculated by me with cultivations of the diphtheria bacillus." Those epidemics, as well as the Worcester outbreak of 1891, and that at Glasgow in 1892, are recorded as having been of bovine origin, but as evidence of the occurrence of bovine diphtheria they are valueless, since in none of them was the presence of the diphtheria bacillus demonstrated in the milk or in the teat or udder lesions. Klein's note on the "cases of causal diphtheria in the cow" at Croydon and at Bishop's Stortford contain no reference to a bacteriological examination. In the Glasgow case the "Hendon streptococcus" was isolated from lymph taken from the teat lesions, whilst experimental inoculation was said to have shown that two varieties of eruption existed, one being true vaccinia, and the other a non-vesicular eruption like that of the Camberwell outbreak, which, it may be said. was not associated with diphtheria. The evidence connecting the cows with the human disease in the Worcester case is on a similar footing.

It was, therefore, purely an assumption to have regarded these diphtheria epidemics as in any way dependent on the teat and udder conditions of the cows, an assumption based apparently solely on the co-existence of these conditions.

Swithinbank and Newman [2] state that the cases in which the *Bacillus diphtheriæ* have actually been isolated from milk are extremely few, and they cite four cases which they state are the only authentic instances of actual detection of the bacillus of diphtheria in ordinary milk with which they have met. Only one of these was associated with an abnormal condition of the teats and udders of cows. Two cows were involved, and both showed papules and ulcers on the teats, covered with dark brown scabs. In one the udder and the secretion of milk were normal, and in the other one posterior quarter was

[1] Local Government Board Report.
[2] "Bacteriology of Milk."

affected with mammitis, the secretion being ropy, purulent-looking, and tinged with blood. Drs. Deans and Todd, who conducted the bacteriological investigation of this case, isolated from the teat lesions bacilli morphologically, culturally, experimentally, and, in their degree of virulence, were indisguishable from those obtained from the throats of the diphtheria patients, and against the effects of which experimental animals were protected by the previous administration of diphtheria antitoxin. They also isolated identical bacilli from the milk of both of the cows, drawn with all antiseptic precautions, notwithstanding that the udder of one appeared to be normal. Experiment showed, however, that the organisms were not the cause of the udder lesions. The cow disease was readily communicable to calves, but the calves were not protected by the previous administration of diphtheria antitoxin, and the conclusion of the investigators was to the effect that the cows were affected with a specific eruptive condition apart from the diphtheritic infection, but that there were no means of determining the manner in which the bacilli of diphtheria obtained access to and multiplied in the lesions of that disease, though they appear to have regarded it as probable that the saliva of the attendants was not free from responsibility.

Deans' and Todd's investigations suggest the possibility of the diphtheria bacillus multiplying in the healthy udder without producing evidence of its presence; in other words, of existing there practically as a saphrophyte. It has yet to be shown whether or not the *B. diphtheriæ* are capable of existing saphrophytically in the healthy udder, but even if they are proved to be capable of such an existence, the fact is not one which would afford support to the bovine diphtheria hypothesis.

I have been completely unable to find any real grounds on which a theory of bovine diphtheria could be based. So far as any part which the cow may play in the dissemination of diphtheria is concerned, the whole weight of evidence points to her acting merely as a convenient stepping-stone, by means of which the bacilli of diphtheria may pass from the human mouth into the milk pail.

In conclusion, the same authorities referred to in connection with scarlet fever may be quoted :—

"Only a person in absolute ignorance regarding the bovine diseases encountered in this country can imagine that diphtheria is a disease occurring naturally in the cow and transmissible from one animal of that species to another."—McFadyean, *Journ. Comp. Path. and Therap.*, vol. iii., p. 166

"Up to the present, it may be said that the evidence forthcoming points in the direction of human rather than bovine infection as the origin of the *B. diphtheriæ* in milk."—Swithinbank and Newman, "Bacteriology of Milk," p. 339.

"The cases recorded in medical literature of the alleged transmission of infection (of diphtheria) . . . are on closer examination reduced to mere assumptions, the forming of which has been due to an entire ignorance of veterinary pathology."—Friedberger and Fröhner, "Veterinary Pathology" (Haye's translation), vol. ii., p. 414.

"The bacillus of diphtheria must come directly or indirectly from the oral cavity of people who are or have been recently attacked with diphtheria. . . . The opinion that was held formerly by some that diphtheria in man could come from a disease of milk cows is entirely erroneous."—Jensen, "Milk Hygiene" (Pearson's translation), p. 114.

### Sore Throat.

It is a matter of common knowledge that a considerable number of cases of sore throat occur in association with the majority of scarlet fever epidemics, whether milk-borne or not, and that the two occur not infrequently in the same household. There is no room for doubt that a close relationship exists between the two. The same holds good for diphtheria. A type of sore throat, however, apparently quite distinct from either of these two diseases, is met with in human beings. It is characterized by congestion and inflammation of the tonsils, sometimes ulceration, enlargement of the cervical glands, fever and general depression, without other evidence of specific infection.

This condition is not notifiable, but its occurrence at times endemically or epidemically has resulted in special inquiries being instituted to trace the source of infection, and as with the two diseases already dealt with, the conclusion has been reached in certain instances in which milk has been shown to be the medium of infection, that abnormal conditions of the cows furnishing the milk have been responsible for its infective properties. Bacteriological evidence in support of this conclusion cannot be found, however, and consequently definite proof of a causal relationship between the human and animal conditions is wanting.

Inflammatory conditions of the udder have been most frequently found in association with these epidemics and have been held responsible for the infectivity of the milk, but eruptive conditions of the

skin of the udder and teats have also, though less frequently, been regarded as the primary cause of the human condition.

Probably it has been the experience of every inspector of dairy cows to find that the secretions from an acutely-inflamed udder have been added to the general stock of milk sometimes for several weeks in the intervals between visits of inspection. Such instances are fortunately uncommon, and almost invariably when the secretions from an inflamed quarter have undergone obvious change, they are rejected by the dairyman independently of the inspector. Given a case of mastitis, however, involving one quarter of the udder only, the custom almost universally prevails of mixing with the general stock of milk that secreted by the healthy quarters, since to all appearances it is perfectly sound. It has recently been shown that micro-organisms indistinguishable from those causing the mammitis may be found almost constantly in the teat canals and in the milk sinuses of the healthy quarters, where they produce no morbid change and no alteration in the milk. It thus follows that if mastitis be a cause of human sore throat, large quantities of milk which are a potential source of infection are distributed and consumed daily.

In subacute catarrhal mammitis there is no apparent change in the milk when first drawn from the udder, but if "set up" for a few hours it shows a marked departure from the normal, and on examination is found to contain streptococci in large numbers. This condition is by no means uncommon, and until the attention of the dairyman is drawn to it the secretions of the affected quarter or quarters are invariably added to and distributed with the milk.

Similarly in eruptive conditions of the skin of the teats. I very much doubt if it is possible to examine a herd of dairy cows of any dimensions without finding several with lesions on the teats, and there are probably few veterinary surgeons who have not had experience of eruptive conditions of the teats of dairy cows communicable from one animal to her neighbour by the hands of the milkers, in which for weeks the milk drawn from animals in all stages of the affection has been distributed in the ordinary way and consumed by human beings without any coincident sore throat or other epidemic.

Experience of this description forces upon us the conviction that if mastitis or eruptive disorders of the udders and teats of dairy cows are capable of producing sore throat amongst the consumers of the milk, the great majority of such cases are not capable of so doing, for if they were at all commonly a cause of sore throat in man, it is obvious that epidemics of that condition would be of very much greater frequency than they are.

The only bacteriological evidence which is available on this question supports this conclusion. Savage[1] has recently carried out a series of investigations into the bacteriology of mastitis and the etiological relationship of that condition to human sore throat. He found that in 75 per cent. of the cases of mastitis which he examined, the causal organism was a streptococcus occurring in long chains, and possessing a very low virulence for guinea-pigs and mice, but inducing, when injected up the teats into the udders of goats, a severe mastitis with long-continued infection of the udder. To this organism he applied the name *Streptococcus mastitidis*. In one of his experiments he injected into the udder of a goat streptococci from a sore teat, with the result that an extremely severe mastitis was produced, with marked general symptoms and long-continued persistence of bacteria in the udder.

Simultaneously, an investigation of the organisms met with in human sore throat was made, and confirming the results of previous investigators, Savage found that two chief types of streptococci prevailed—the *S. pyogenes* and the *S. anginosus*—the latter being the more common, and corresponding to the streptococcus which exists in large numbers in the throats in scarlet fever.

He showed that culturally and morphologically the *S. anginosus* is indistinguishable from the *S. mastatidis*, that experimentally it possesses considerable virulence for mice, and that it is incapable of originating mastitis in goats. The effects of the *S. anginosus* when inoculated into the udder of goats thus afforded a means of differentiating it from the *S. mastitidis*, and Savage suggested as a practical point that the best way to determine if outbreaks of human disease were spread by streptococci associated with inflammatory cow conditions was to isolate the streptococci from the human disease and see if they would cause mastitis in goats by inoculation up the teats. If they were of milk origin they should produce mastitis in goats. These results suggested to Savage that the *S. mastitidis* is not a cause of human disease.

Bacteriological evidence, therefore, so far as it is available, does not support the belief that human sore throat has its origin in mastitis or teat lesions of the cow. But the evidence is not sufficient to permit of the complete exclusion of these conditions as causes of human sore throat. The many investigations into the bacteriology of mastitis in cows have shown that different varieties of bacteria may be the cause

[1] Local Government Board Reports, 1906-07 and 1907-08.

of that condition in cows. Streptococci undoubtedly are most frequently responsible, and whilst one type predominates, several other varieties have been distinguished. Staphylococci are less frequently found, and are usually associated with the milder forms of mastitis. Still less frequently bacilli, usually of the coli type, are met with. Not uncommonly several different types of organisms co-exist in the secretions from an inflamed udder, making it difficult to determine which has been responsible for the morbid conditions. (Specific conditions, as tuberculosis, are not at present under consideration.) It is conceivable that one or more of these types of bacteria may be capable of producing sore throat in man, but I have seen no report of a sore throat epidemic which had its origin in infection through milk, in which the facts brought to light in the course of the investigations have established an etiological relationship between the human conditions and either mastitis or sore teats in cows.

There is another point which is of importance in this connection. Staphylococci and streptococci, bearing a close resemblance to those which are a cause of mastitis except in their pathogenicity for animals, may be found in the teat canals and milk sinuses of perfectly healthy udders. Their numbers vary within wide limits, but they are rarely absent. Since they are found with such great frequency very few can, and probably none do, possess pathogenic properties for man. They appear to be purely saphrophytic. The demonstration, therefore, in the throats of human patients infected by milk, of bacteria culturally and morphologically similar to those found in the udders, whether apparently healthy or not, of cows on the premises from which the infected milk is obtained, falls very considerably short of proof of an etiological relationship between the human and animal conditions.

Whilst the experience of veterinary surgeons inspecting dairy cows and the bacteriological evidence available do not support the view that a relationship exists between human sore throat and morbid conditions of the udders and teats of dairy cows, the question is one which can only be definitely settled by time and the most careful investigations of human epidemics carried out by highly trained observers as opportunity offers. But whether or not it is ultimately proved that an etiological relationship sometimes exists, all will agree that the milk of cows affected with mastitis in any form, or with teat conditions in which discharges or material from the teat lesions are liable to find their way into the milkpail, should not under any circumstances be permitted to be sold and used for human food.

## A FEVER IN HORSES SIMULATING HORSE-SICKNESS.[1]

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Before the war, and repeatedly since then, I have come across a fever in horses which simulates horse-sickness in a certain respect, but differs from it inasmuch as no horse succumbs. I called it "ephemeral fever" to indicate its mild character. It needs hardly to be said that, besides this fever, there are still others met with in horses, also excluding biliary fever and horse-sickness, and which may be specific, but they have not yet formed the subject of such a close study as that under discussion, which was met with under peculiar circumstances. The term "fever," in its restricted sense, means only a symptom of a disease, that is, the high temperature which indicates a reaction of the animal's system to some obnoxious influence. "Fever," in its wider sense, may also stand for the word "disease," as, for instance, in the term "biliary fever." In speaking of "ephemeral" fever, I use the term in this, its wider sense, meaning a disease, and indicating at the same time that the fever is the main symptom, other characteristics which might suggest another name being absent. "Ephemeral fever" therefore indicates a certain morbid condition of horses which cannot be recognized with accuracy in the first instance, and which has practically but little economic importance.

*Cause of the Disease.*—Since bacteria and protozoa cause infections which are accompanied by fever, we naturally suspect in all fevers the presence of organisms. The microscopic examination of blood of horses during the acute stage of this fever gave negative results. The blood spread out on different culture media with the object of growing the supposed micro-organism, also gave negative results, notwithstanding that large quantities were used both for aerobic and anaerobic cultures. This proves that the disease is not caused by bacteria, which, had they been present in the blood, if only in small numbers, could not have escaped observation in this way. The fever, however, is transmissible by the inoculation of blood of an animal suffering from it, and during the whole time the disease lasts. A small quantity of blood is sufficient to reproduce it in the injected animal after both subcutaneous and intrajugular injections. A further peculiarity is that blood which is conserved with preservatives also retains the virulency for a considerable length of time.

[1] From the *Transvaal Agricultural Journal.*

Further, the serum which has been separated from infective blood transmits the disease. Finally, if such serum be filtered through a Berkefeld filter, which retains the usual visible micro-organism, it is observed that the filtered liquid still reproduces the fever, making it apparent that the virus belongs to the group of ultra-visible micro-organisms which cannot yet be detected even by the highest magnification of our present microscopes. These facts give the cause of the fever a striking resemblance to horse-sickness, even in details. The question had therefore to be taken into consideration whether it could not be identical with horse-sickness, being only of a milder character and representing some peculiar strain, as we know by this time that strains vary greatly in virulency, a fact which makes it so difficult to obtain immunity against this disease. For this reason it was considered necessary to prove its identity, or otherwise, with horse-sickness.

### Differentiation of Horse-sickness and Ephermeral Fever.

One of the characteristics of horse-sickness is the incubation time, that is, the period which elapses between the infection of the animal and the first rise of temperature. It is true that it varies in length, and that in the acute form it is shorter than in the sub-acute forms, but it has an average length of eight days. The ephemeral fever has a shorter incubation time, averaging almost regularly five days; it may be shorter than this, but is very rarely longer. In horse-sickness, as a rule, the fever rises gradually, increasing daily one or more degrees until it has reached the climax when either the animal collapses and dies suddenly, or, just as the fever has been rising by degrees, it descends again by degrees, and the descending curve resembles very much the ascending one. This period of fever in the recovering animal lasts very rarely less than a week, and usually ten days. In ephemeral fever we notice something similar, but the rise of the temperature is more abrupt, and the climax is reached within a much shorter time. The fever does not last long and soon descends, so that the whole length of the fever does not average more than about four or five days. The acute ephemeral fever therefore does not resemble in its curve the acute fever of horse-sickness. There are, however, exceptions to this general rule, where the fever curve resembles that of horse-sickness to such an extent that, even by more close comparison of the two, a diagnosis would be impossible. In horse-sickness a sudden dropping of the temperature invariably means the approach of death, but such is not the case in ephemeral

fever, where the temperature can drop from its climax to normal within one day, and yet we notice nothing which would resemble the collapse in horse-sickness.

The ephemeral fever is accompanied by an increase in the activity of the heart. During its sudden rise, and whilst it is high, the number of beats is increased. In acute cases this increase numbers eighty beats and more, and at the same time the quality of the pulse is somewhat altered, the individual pulsation being hurried and soft, so that at a given moment, and considering the high fever, one is inclined to make a bad prognosis. In addition to this a slight increase in the number of the respirations is occasionally noticed; the character of these, however, is never such as to lead one to suspect the presence of horse-sickness. In examining the mucous membrane of the eyes, occasionally a slight injection of the membrane, or diffuse reddening is observed. The horse may even be noticed to be off his feed, standing somewhat drowsily at the manger, and will be found to be less spirited than usual. The presence of these symptoms, of course does not allow of an immediate definite diagnosis as they may initiate many diseases which can only be diagnosed by the course of further events. For differential diagnostic purposes the quality of the pulse may be taken into consideration. In a case of horse-sickness, with such a rapid pulse as in ephemeral fever, we usually get a rapid respiration at the same time, which leaves but little doubt about the character of the disease, or the symptoms of dikkop are present, which also is typical. The bilious fever in horses begins somewhat similarly, also with a high temperature and a frequent pulse, but on examination of the mucous membrane of the eye we find almost regularly the jaundiced condition which is typical of this disease, or if not yet present, it develops at least within twenty-four hours and becomes so pronounced that the disease can be diagnosed. Although we now differentiate this disease from horse-sickness, which it resembles in many ways, yet the absolute certainty of their dual nature we obtained only by experiment. Theoretically speaking, the characters mentioned were not sufficient to settle the question of the identity or non-identity, because we know certain horse-sickness strains have certain peculiarities both in the length of incubation time and in the length of the disease itself. More decisive points had to be looked for to demonstrate their non-identity. We know of horse-sickness that when we inoculate a large quantity of virulent blood into the jugular vein of a susceptible animal, that this disease appears without an incubation

time, and the course is so rapid that the animal invariably dies within a very few days. The same experiment with ephemeral fever, whereby the blood of one horse suffering from the fever was transfused into a susceptible one, in quantities of 5 litres, produced nothing more nor less than the fever. It was impossible to kill a horse with this fever.

*The Immunity Reactions.*—Before entering into the question which may be considered to be the most important—whether an animal which has recovered from this ephemeral fever has obtained immunity against horse-sickness—it has to be stated first that an animal immunized against ephemeral fever remains immune against a subsequent injection of the same strain. Undoubtedly a fever caused by the smallest quantity of virus protects against a subsequent inoculation of a large amount of virus of the same strain. We may say the immunity is complete against the particular virus which has formed the subject of the experiment. Approaching the question of the immunity against horse-sickness, a fact must be remembered to which I have referred in the past in various articles on immunity in horses and mules, viz., that an immune horse or mule may break down in immunity when we use a strain of virus different to that which has been used for the immunization of such an animal. It is true that there are horses and mules for which the immunity obtained by one strain is sufficient to protect against any other subsequent inoculation of different strains, but there are also horses and mules which react not only once, but twice, and even more times, to subsequent inoculations. In order to emphasize this, I have pointed out cases showing that horses and mules can, in a period of a few weeks, suffer three times from dikkop. In scientific research use is generally made of the principle that immunity obtained by the injection of micro-organisms protecting against the injection of a similar micro-organism of different origin indicates that both are identical. This principle has lately been applied in the identification of the various trypanosoma diseases. It does not hold in horse-sickness as we have just demonstrated above. This point has to be taken into consideration, and a breakdown in immunity does not necessarily exclude the non-identity of two micro-organisms, or of two diseases. Experiments on those lines are then of value when they are undertaken on a fairly large scale; coincidences may then be excluded by statistics. This was the case with our ephemeral fever, where subcutaneous and intravenous injections were made use of; the virus was injected in small and large quan-

tities, and the animals immune to ephemeral fever were then submitted to test by horse-sickness virus, of which the various strains were used. In no instance was there the slightest indication of any immunity obtained by the ephemeral fever, and the horses contracted and suffered from horse-sickness just as horses known to be highly susceptible to this disease. The reverse experiments were also carried out. Horses immune to various strains of horse-sickness were injected with small doses of ephemeral fever virus, and in every instance this fever appeared, and in no way differed from the fever met with in the susceptible animal injected in the same way. Therefore no doubt can be left that these two diseases are of different nature; the one a very harmful disease, and the other harmless.

*Origin of the Disease.*—It will be interesting to note how we came across this ephemeral fever. I may state that we met it in some instances occurring spontaneously in horses which were running outside during the day, and which were placed in a stable at night. It is the routine procedure of the laboratory to take the temperatures of all animals twice a day—in the morning and evening — and whenever it is found that any fever is present, the horse is thoroughly examined and the blood immediately placed under the microscope. When a diagnosis is not possible, inoculation experiments are then undertaken. Further, we came across it in a mule suffering from horse-sickness; the animal was recovering from dikkop, and for the purpose of obtaining a fresh horse-sickness virus, it was bled and the blood injected into another animal. It happened that the doses of virus which were injected were not sufficient to produce horse-sickness, but this ephemeral fever described. When the same strain of horse-sickness virus from another animal was used, the same animal contracted horse-sickness, proving that the previous mild reaction was not identical with this disease. This experiment indicates that more than one organism may be present in one and the same animal at the same time. This particular association we have difficulty in explaining at the present time otherwise than by accepting a mere coincidence. Further, we came across the disease in horses which were exposed during the night in a locality where horse-sickness was known to exist; the exposure was made for the purpose of giving the animals horse-sickness in the natural manner, aud one of them contracted this fever. It was somewhat long in reaction, resembling the horse-sickness reaction more than any other, but there was no doubt that it was this ephemeral fever and subsequent inoculation supported the diagnosis. This one experiment threw

some light on the manner in which the disease may be contracted. I have but little hesitation in stating that it must be contracted in somewhat a similar way to horse-sickness. Although I have proved, or think to have proved, that ephemeral fever and horse-sickness are quite different diseases, yet there may have existed at one time a certain relationship between the two. If we believe in the evolution of organic life, we can apply the principle to the micro-organisms of diseases as well, and accept the view that the differentiation took place at an early period, one developing into the fatal horse-sickness and the other one into the mild ephemeral fever. Of course, these are theories, but they permit of an understanding of certain phenomena which are difficult to explain.

I have indicated that we obtained the ephemeral fever from different sources, and one may rightly bring forward here the question whether all these various fevers were identical with each other, or whether the various ephemeral fevers represent as many entities of their own. Accordingly the immunity test was applied again, that is to say, the immunity obtained from the one source was tested against that of another source. Now we noticed exactly a similar thing to that to which we drew attention before occurring in horse-sickness. There does exist an immunity, generally speaking, that is to say, the majority of animals injected with the virus of one source were protected against the one from the other source. There were exceptions to this rule, and some horses reacted to both injections, thus proving that not every horse has obtained immunity by the one injection. Finally, the question arose, always having in mind a difference in virulency, and the difference in the susceptibility of the horses, whether animals protected against ephemeral fevers of different sources would be protected against horse-sickness. All experiments in this respect failed again.

The practical conclusion from this short *exposé* may be considered to lie in the fact that animals may suffer from a very acute and high fever without this fever interfering at all with the health. It is generally believed that a high fever indicates a serious state of affairs in the economy of an animal. Speaking generally, this is true, because most diseases which end with death are accompanied by high fever, but the reverse does not hold good. Not every high fever means death, and accordingly it shows that there is a difference between the cause of the fever and the cause of death. We differentiate between a pyrogenous[1] and a toxic[2] agency. The former acts

[1] Producing heat. [2] Poisonous.

principally on the nervous centres regulating the body temperature, and the latter attacks and disturbs the vital parts.

The treatment of the fever, as such, is therefore not so necessary as it is generally thought to be, and if by the administration of drugs it can be reduced, it does not follow that the toxic effect is done away with. At the same time, if that was the case, the treatment of all diseases would be a comparatively easy thing, because we are to a great extent able to reduce fever, but nevertheless the animals die.

The second practical inference from these observations is that at the time when horse-sickness occurs, fevers are occasionally met with in horses which laymen consider to be horse-sickness, and when they observe the symptoms which we have described, and the animal has recovered, they are under the impression that the animal is salted against this disease. Unfortunately we are not yet in the position to differentiate in practice between the mild attack of horse-sickness and the ephemeral fever, and it is therefore quite natural that not only laymen, but experts, may come to the conclusion that certain fevers are cases of horse-sickness when indeed they are not of this nature.

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## SWINE ERYSIPELAS.[1]

This disease may be defined as a contagious disease of swine caused by the bacillus of swine erysipelas.

### Prevalence.

The investigations which have been conducted in connection with swine fever have shown that swine erysipelas, particularly in its milder forms, frequently affects pigs in Great Britain, and that in a certain proportion of cases it is the cause of death.

During the last three years records have been kept by the veterinary officers of the Board regarding outbreaks of disease which were reported as suspected swine fever and afterwards found by the visiting veterinary surgeon to be swine erysipelas. These records, which extend to 1,200 such outbreaks annually, show that at least one pig died in each case.

This figure, however, cannot be taken as nearly representing the annual number of outbreaks of swine erysipelas, because in most cases the diseases assumes a mild form, and there must be many other out-

[1] Leaflet issued by the Board of Agriculture and Fisheries.

breaks in which a pig has died without raising a suspicion of swine fever, in which case the fatality would not be reported to the Board. The disease is most frequently met with in fat pigs—that is to say, at a somewhat later age than that at which they are most usually attacked by swine fever.

### Symptoms.

*Acute Cases.*—In acute cases of swine erysipelas the animals show the usual signs of severe illness in the pig—viz., rise of temperature, shivering, loss of appetite, and vomiting. In such cases a fatal termination may take place in twenty-four to forty-eight hours, but the animals frequently live much longer. In the less acute cases a red patchy eruption, from which the disease gets its name (erysipelas), appears on the buttocks, thighs, body, and ears.

The breathing is very rapid, and the swine stagger about when made to walk. Ultimately they lie prostrate in the litter and die comatozed.

*Mild Cases.*—In mild cases the general symptoms are not marked; the swine appear to be out of sorts, and show the usual skin eruption, which is sometimes called nettle rash.

Animals which have apparently passed through the acute stages of the disease may remain unthrifty for a long time. Sometimes they die suddenly from disease of the heart, which is not an uncommon sequel of the disease. In other cases they present symptoms of lameness due to trouble in the joints.

The skin is discoloured by livid patches as in swine fever, but sometimes the only symptoms shown are those of nettle rash. The bacillus apparently can flourish for a long time outside the bodies of animals, so that once the disease is introduced into insanitary styes the infection tends to remain there. For some reason, however, which is ill understood, the disease may assume a very mild form for a time, then burst out acutely. In Great Britain the acute forms have been observed particularly in the warm months.

*Post mortem.*—The membranes of the stomach and intestines show red patches and are often swollen. The intestinal glands on the membrane are red and enlarged; sometimes the surface over these glands is abraded, but the distinct ulcer of swine fever is never seen. The lymphatic glands throughout the body are swollen and red. The spleen is often enlarged.

The membranous coverings of the lungs and hearts show red spots, and sometimes water is present in the chest and heart sac.

The lungs are congested.

In the chronic form the tissues around the opening between the chambers of the heart, particularly on the left side, are frequently thickened and rough—that is to say, endocarditis is present.

### Prevention and Remedies.

This is a disease against which several methods of protective inoculation have been directed. At the present day it is customary to employ either (1) a preventive serum obtained from horses which have been highly immunized by the injection of large quantities of pure cultures of the bacillus of swine erysipelas, or (2) a combination of specified doses of preventive serum and pure cultures of the bacillus.

The immunity conferred by the serum alone begins immediately, but it lasts for little more than ten days. Apparently, however, it exerts a protective action even when used in the initial stages of infection. The immunity conferred by inoculating with both serum and pure culture lasts a much longer time, probably six months and even longer. Leclainche, who has been foremost in elaborating this method of protective inoculation, advises that where the disease has already broken out, the pigs should receive a preliminary injection of serum, 10 to 20 c.c. according to weight. This, he states, greatly reduces the number of accidents consecutive to vaccination proper—that is to say, with the combination of pure culture and serum which is performed about ten days afterwards. The vaccination proper consists of two operations. The first is performed with a mixture (made on the spot) of serum—1 c.c. per 20 lb. live weight, with a minimum dose of 5 c.c. and a maximum of 10 c.c.—and ·8 c.c. of a pure culture. Twelve days later the second operation is performed, when the animal receives ·8 c.c. of a pure culture without any serum. The materials are injected subcutaneously, either at the base of the ears or inside the thighs, During a period of eighteen months ending November, 1901, Leclainche had under observation 24,000 pigs which had been inoculated by his method; about one-half of these were treated by a preliminary injection of serum alone. Not a single accident was recorded.

Lorenz has reported observations on 22,161 pigs which were inoculated in Eastern Prussia by the combined method (serum and culture), and 3,831 of these pigs were on farms on which the disease had already broken out. In the latter there were no fresh cases of swine erysipelas after inoculation; 50 per cent. of recoveries were recorded in sick animals after the injection of serum alone one to four doses). Nettle

rash, which is a mild form of swine erysipelas, occurred in ·04 per cent. of the inoculated animals. The disease disappeared from the farms after inoculation was adopted, whereas it had appeared at regular intervals before that time.

RECOMMENDATIONS.

(1) It is not advisable to resort to inoculation of pigs on non-infected premises unless the circumstances are such that owing to the proximity of acute outbreaks it appears practically impossible to prevent the disease being introduced by methods of rigorous isolation, because the operation might possibly be the means of infecting the premises.

(2) Should the disease appear, however, all the pigs should, with the least possible delay, receive a dose of serum, and those in which the temperature is normal should be removed to non-infected styes on the same premises, if this be practicable. Ten days afterwards the vaccination proper may be practised after the method of Leclainche (serum and virus, then virus alone) on those animals still showing a normal temperature. The pigs with high temperatures should be returned to the infected styes, and if their value warrants it, they should be treated by injections of serum alone. On no account should they receive the culture. If it be found impossible to separate the sick from the healthy, the operations should be carried on in the infected styes.

(3) Although this disease can to a large extent be successfully combated by inoculation, it must not be thought that measures of isolation and sanitation can be dispensed with. While the outbreak lasts no new pigs should be brought in, and none should leave the premises except for slaughter under the most rigorous precautions against the disease being conveyed to other premises. If a pig-owner finds that the disease reappears annually on his premises, he should resort annually to preventive inoculation, timing the operation so as to have his animals immunized before the season of greatest activity. He should also remember that the complete eradication of the disease from his premises will be greatly facilitated by keeping his pigs in styes which can be properly disinfected.

Although swine erysipelas can hardly be regarded as a very fatal disease of pigs, in Great Britain at least, the Board have been informed that it often interferes materially with the marketing of pigs, since it frequently attacks them and causes considerable emaciation close to the time when they are expected to be ready for market.

In such cases owners have been advised to immunize their pigs by methods of inoculation about three months before they are expecting them to be ready for market. From information received from those who have put this advice into practice, it would appear that the adoption of preventive inoculation has given excellent results.

As swine erysipelas is not a disease notification of which is required by Order of the Board, it should be borne in mind that a pig suffering from that disease may also be affected with swine fever, and that the Swine Fever Order of 1908 requires that every person having in his possession or under his charge a pig affected with, or suspected of, swine fever shall give notice to the police. In this connection attention is called to the "Notice to Pig Owners" issued by the Board.

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## ON THE EMPLOYMENT OF A THREE PER CENT. FORMALIN SOLUTION.

By Veterinary-Surgeon PAMPERIN.

In a riding horse several hard lumps as big as peas formed, lying thickly together in the saddle neighbourhood, and eventually developing into three nodes, each as big as half a pigeon's egg, I immediately tried to remove one of the lumps surgically. I found, however, that it was situated rather deeply in the subcutaneous connective tissue, so I desisted, and endeavoured to remove all three nodes with the above-mentioned solution. The well-known deep effect of formalin induced me to try it. After shaving the hair away in the neighbourhood of the lumps and over them, and disinfecting, I made two incisions at right angles down to the roots of the nodes and divided them into two halves. After cessation of hæmorrhage I dropped some of the solution into the incisions, and let the horse stand without being rugged. On the following day a few drops more were put in.

After the fourth day the divided knots began to loosen, and on the eighth day, up to which time a few drops daily had been introduced, I could remove the lumps piecemeal with the forceps. In the gaps left, which were carefully cleansed daily, I poured iodoform collodion and covered them with wadding. After further fourteen days the holes had closed up, and the horse was discharged cured.

I also used this means with good success on a dog with cartilaginous swelling, as big as a two-mark piece, on the right elbow-joint. I divided the swelling, poured in formalin solution, and bandaged the the place. Here also the knot could be taken away in pieces on the fourth day. The resulting wound was covered with iodoform collodion, and after twelve days' treatment a cure was effected.

No return of the swellings has been noted in either case, so a 3 per cent. formalin solution may be recommended.

(*Zeitschrift für Veterinärkunde.*)

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# Clinical Articles.

## FOREIGN BODY IN THE HEART OF A CALF.

By Wm. MOODIE, M.R.C.V.S.

*Rothesay.*

I am sending you the photo of what is probably a unique case, showing the left ventricle of the heart opened, and embedded in the wall a piece of wire 2 in. long and hooked, the bent part being at the apex and ¾ in. in length. This was taken from a 7 weeks old calf in good condition, and my attention was called to it by Mr. Squair, butcher, who, on killing the calf and when disposing of the heart, in

Heart cut open, showing Wire in the Myocardium.

cutting it up, as shown, his knife was carried right along the wire. There was no inflammation of the pericardium or endocardium, nor of the fleshy substance of the heart; externally and internally the mucous lining was perfectly healthy and natural, and the wire lay encased in a thin fibrous covering—as the butcher expressed it, like a meërshaum pipe in its case.

How and when did the wire get there? The calf did not swallow it; when did the cow, and how did it travel to the position shown in the photo? I leave your readers to speculate.

The calf never showed any signs of ill-health during life.

## A CASE OF *POST-PARTUM* TETANUS IN A COW.

By NORMAN MEYERS, L.V.Sc.
*Caulfield, Melbourne.*

On Sunday, July 17, I received a telephone call to attend a cow, which, from the meagre message given, I presumed to be one of those varied cases associated with the act of parturition. On examining the animal, however, it took me but a short time to decide on it being a case of tetanus, and as such are comparatively rarely seen in bovines, the following clinical observations may be of interest:—

*History.*—The cow had calved some seven or eight days previously, and during the next twenty-four hours everything had appeared to be going on satisfactorily. From this period onwards, however, the animal evinced little inclination to take any food, and for the following few days the symptoms were those usually associated with "retention of the fœtal membranes."

Home remedies were given in the form of drenches, which were taken readily by the animal. On the Friday previous to my arrival the uterus was irrigated by means of the inevitable garden hose, with the result that a slight improvement was shown in the patient's condition. By Saturday afternoon the patient had again become worse, and through either lack of inclination or inability would partake of neither food nor water.

*Symptoms.*—The patient was standing in the corner of a small paddock, and on my approaching her showed signs of very marked timidity, which became visibly accentuated on handling. The head and neck were held in a rigid position, and at first sight this, perhaps, was the most marked feature of the case.

The picture when she moved was almost a grotesque one, for with stilty short steps forward she showed no liking to move either to the right or left, appearing all the time to be in danger of falling. When she did attempt to turn, the hind legs acted as a kind of pivot, on which the fore part of the animal was able to bring itself to the required position.

The masticating muscles were in a state of tonic contraction, so that prehension of food was impossible, and all my efforts to part the lower from the upper jaw were futile. Tenacious frothy material was adherent to the lips. The ears had lost their mobility, being held in a forward and outward direction. The eyes, though fixed and staring, showed no retraction, nor was there any protrusion of the membrana nictitans over the cornea. The neck muscles on palpa-

tion seemed as hard as a board, while those of the lumbar and abdominal regions were tense, so that a very "tucked up" appearance was presented. The vertebral column showed no alteration of curvature, and the tail, contrary to what is generally seen in horses, was hanging in the usual way.

The nostrils remained dilated, and although the respirations were short and shallow, yet they were louder and faster than normal, and increased to a marked extent on the slightest excitement.

Careful examination failed to locate any external wound whereby infection may have taken place, and although it is quite possible that it may have been associated with the act of parturition, yet the fact of no tetanic symptoms appearing until after the uterus had been irrigated inclines me to the belief that the garden hose was the germ carrier, while an abraded surface on the uterus caused by rough manipulation formed a favourable spot for the growth of the bacilli. Assuming this to be correct, the period of incubation of the tetanus bacillus in this case would be one of from twenty-four to thirty-six hours.

As the case was a particularly depressing one to the owner, and partly for economic reasons, no treatment was attempted.

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## A CASE OF SHOULDER LAMENESS.

By J. J. O'CONNOR, M.R.C.V.S.

*Professor in the Royal Veterinary College, Dublin.*

*Subject.*—A nice looking, fat, roan pony, aged 4, by a trotting stallion out of an Iceland pony.

*History.*—Had been very lame on the near fore limb for twelve months, although doing no work and being well cared for.

*Symptoms.*—Those of severe shoulder lameness. When in the stable the animal was very uneasy on the limb, constantly shifting it. At a walk there was great difficulty in advancing the limb, and at a trot she went on three legs. When the shoulder joint was passively flexed great pain was evinced. The external tendon of the infraspinatus, which passes over the internal tuberosity of the humerus, felt very hard, and when manipulated gave the sensation of a loose piece of bone, whose surfaces had become smooth, like a partial fracture of the tuberosity. After trying a blister without avail, I cut down on this tendon and found that there was no loose piece of bone. I removed a portion of the tendon at its insertion. Immediately afterwards the gait was greatly improved, and in the

course of the next few days the animal went almost perfectly sound, using the limb with great freedom, and standing well on it in the stable. But, after about a fortnight, when the wound was almost healed, she became lame again and eventually got as bad as before, having been at grass all the time. After some months the pony was brought to me again, and I now cut the fibres of the post spinatus where it seemed tense above the point where I did tenectomy, and this having no immediate good effect I line fired the shoulder joint. The pony was then sent to the country for the winter, and I did not see her until about eight months afterwards, when she was going almost sound, showing good action. It was a strange case of marked improvement after being extremely lame for about two years.

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## POLL EVIL.

By W. CARGILL PATRICK, F.R.C.V.S.

*Mullingar, Ireland.*

As the above condition has claimed a good deal of attention lately, amongst City practitioners in particular, I thought the following remarks and accompanying photographs might prove of some little interest:—

The subject was a light mare, aged 6, who had fallen under a load and got her head under the shaft during her struggles to regain the standing position. After being released she got up unaided, and was apparently all right beyond what seemed temporary bruising, yet was allowed a few days rest to get over any stiffness that remained.

Some time later, probably a few weeks at most, a swelling began to manifest itself at the seat of poll evil, as well as some difficulty in rotating the head. This was treated with cooling applications and a blister later; but as no improvement was noted, it was decided to operate, although no distinct fluctuation or pointing could be felt. Accordingly, the enlargement was cut into, and a fair amount of deep-seated pus given vent to, disclosing a new growth of bone at the base, in the form of osteophytes on the ala or wing of the atlas, but at that time we considered it did not extend to the neural canal, and consequently it was removed as well as possible, leaving only a roughened surface discernible on careful examination with the finger afterwards. The resulting wound was treated in the usual way, and the patient, making satisfactory progress, was sent home five weeks from date of operation with the wound looking quite healthy, and uninterrupted repair seemed assured. A week later we were asked to see the mare

Lateral view of Atlas in Mr. Patrick's Case of Poll-Evil.

Anterior view of Atlas in Mr. Patrick's Case of Poll-Evil.

again, as she had some difficulty in moving about her box, yet appeared healthy otherwise. However, on visiting the case, well-marked symptoms of inco-ordination were apparent, and a slight enlargement or fulness over right side of the atlas. As the pressure symptoms became more pronounced, in spite of physicking and deobstruent treatment, we concluded that the bony growths must be impinging upon the spinal cord and further treatment hopeless. She was therefore slaughtered.

On *post-mortem* examination we found the atlas had a large osteophyte on the right wing, and overhanging the neural canal to some extent. The right pedicle and wing were also much thickened, the articular surfaces or oblique processes of that side were slightly constricted from above to below, and a distinct gutter from the anterior foraminæ extending to the seat (or normal position) of the posterior foramen was present, the latter being entirely absent. The un-named oramen on the wing of the atlas was nearly four times larger than normal.

The changes present suggest the following queries: Was there any abnormality of bone present prior to the date of injury, and if not, what is the average period of time necessary for such pronounced changes to take place? This applies chiefly to the gutter, which evidently accommodated a blood-vessel, and to the obliteration of posterior foramen.

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## CASE OF FRACTURE OF THE FOURTH CERVICAL VERTEBRA.

By A. W. M. SWANSTON, M.R.C.V.S.
*Captain, Army Veterinary Corps, Colchester.*

Horse No. 12999, of 99th Battery, R.F.A., was admitted into hospital with reported "colic" on July 9 last. All symptoms of this passed off, however, after treatment, and the horse then appeared in a dazed condition. Soon after the animal was admitted it was reported that previous to the attack of colic it had reared up and fallen backwards on to its poll. When approached now by anybody it got very excited, reared up, and on the day of admittance it fell once on to its off-side.

The treatment consisted in keeping the horse very quiet in a loose box, and under constant observation; ordinary diet.

On July 10 no sign of colic was visible, but the horse appeared very uncomfortable, though it fed and drank well. It seemed rather

timid, however, and if anyone went into the box it invariably turned away towards a corner. There was no rise of temperature, and the pulse and respirations were barely altered.

On July 13 the horse was observed to have fits; it turned round and round in circles, always from right to left. On July 14 a very painful swelling was noticed on the right side of the neck. Up till now the horse had continued to feed and drink well; it ate everything in the shape of food that was given to it, and it drank quite a normal quantity of water.

About 11.30 p.m. on July 17 the orderly farrier came to my house and reported that the horse was down and "in a bad way," to use his his own words. On arrival at the sick lines I found the horse in much pain. I gave it 1 oz. of choral hydrate, and made it as comfortable as possible with bedding in sacks. The next morning the horse was up, and as the effects of the choral worked off it took its food and water during the day. It had one fit about noon, and two more during the afternoon and evening.

On July 19 the horse had several fits during the day, always turning in the same direction—*i.e.*, from right to left. The horse appeared to get its neck twisted to the left, and was not able to get it back again, but when it did succeed in doing so, the fit passed off.

On July 20 at early morning stables the horse was found to be lying down. It gradually lost consciousness, but death did not actually take place until after 2 p.m. on Friday, July 21.

*Post-mortem* examination showed fracture of the left transverse process (throughout its whole length), and the posterior division of the right process of the fourth cervical vertebra.

This case is of interest inasmuch as the horse lived from July 9 till July 21.

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## "AN OBSCURE CASE." WANTED—A DIAGNOSIS!

BY AUG. LEANING, M.R.C.V.S.

*Captain, Army Veterinary Corps, Woolwich.*

A REMOUNT was awaiting discharge from the sick lines, after being affected with catarrh for twenty-six days. The horse had had two inoculations with Rotterdam anti-strangles serum. The history of the case is as follows:—

The horse was reported off-feed; temperature 105° F.; respirations not hurried; pulse fairly strong; and no sign of catarrh. The following day the temperature was 104·2° F., and there was no catarrh

during the morning. At midday there was a profuse discharge from both nostrils. This was mixed with blood, and a thick ropy material as if the Schneiderian membrane was sloughing away. Later in the day the breathing was stertorous, discharge thicker, and the nasal chambers nearly blocked. At 7 p.m., as the horse was getting so distressed and there appeared every probability of suffocation, tracheotomy was performed. The horse was much easier afterwards and fed a little, but death occurred a few hours later.

*Post-mortem Examination.*—The epiglottis and glottis were very congested. Turbinated bones on both sides were full of blood clots, giving them an appearance almost like liver. They were quite close together, and occluded the air passage. The mucous membrane had sloughed away. The bones appeared to be more of a cartilaginous than bony nature. On section they were of a dark red colour, and had the appearance of liver in an advanced state of cirrhosis. The Schneiderian membrane was of a dark red colour. The alae of both nostrils were considerably thickened and congested. The trachea was normal. Both lungs were in a state of acute pulmonary congestion, and contained numerous hæmorrhagic areas. The right lung had a wedge-shaped infarct extending from the surface inwards for two inches. Heart: The myocardium was inflamed and showed numerous ecchymoses in both ventricles. There were numerous adhesions between the liver and diaphragm, the remains of an old-standing perihepatitis.

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## CASE OF SEPARATION OF EPIDIDYMIS FROM TESTICLE.

By W. STAPLEY, M.D., M.R.C.V.S.

*Professor of Surgery in the Veterinary School of the University of Melbourne, Australia.*

Being asked to place on record a bungling operation I did on a horse with a hidden testicle, I do so to prevent further mistakes of a similar nature.

A four-year-old horse had been castrated on the right side. The left testicle could not be found by the castrator.

Bimanual examination was negative. Incision over the external ring at once brought to light a mass beneath the cremaster, which appeared as a fully-developed epididymis and a greatly atrophied testicle. It was removed. The mass was submitted to an expert histologist, who by frozen section said it was undeveloped testicle, but

he asked for time to prepare a proper section. After a delay of ten days, no report being received from the histologist, I prepared to send the animal home—some 150 miles distant. Luckily the animal charged a stable boy, got loose, careered through the heart of Melbourne, and was astray at the time he was booked as a passenger on a steamboat. That night the histologist reported "the tissue is certainly epididymis, and not testicle." On recovery of the animal I again operated, and removed a large flabby testicle that was free in the belly, hanging by a long mesentery from the loin.

The case is recorded because of the wide separation of tissues normally associated. An investigation of fœtal horses reveals at once the possibility of such a wide separation, and some marsupials, such as the opossum, show normally a separation of testicle from epididymis. As far as I can ascertain, there is no record in human surgery of a similar separation of testicle from epididymis in man. I believe this condition in the horse has been previously reported; it is, however, such an important surgical condition that I felt a brief record of this case may serve to keep our memories clear on the fact that such separations occur, and should be looked for when operating on monorchid or on cryptorchid cases.

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## FATAL INTERNAL HÆMORRHAGE AFTER CASTRATION OF A RIG.

By J. J. O'CONNOR, M.R.C.V.S.

*Professor in the Royal Veterinary College, Dublin.*

Out of a large number of rigs that I have operated upon, I had the good fortune not to lose one until recently, when I lost one from internal hæmorrhage.

*Subject.*—A strong cart horse, aged 3, in very good but soft condition.

*Operation.*—Cast and chloroformed, fixed in the dorsal position, took the usual precautions, removed the large testicle which was in the left scrotum by torsion, no bleeding resulting from the end of the cord; explored the right inguinal canal, found there the vas deferens in a fibrous sheath, opened the sheath and tried to draw out the testicle through this opening but failed, even after dilating it somewhat by means of a long forceps. I then made an opening through the anterior wall of the inguinal canal, higher and further outwards than the internal inguinal ring, by means of a blunt perforator, inserted my two fingers into the opening and found the

testicle, which was fairly large, hard, and fibrous, drew the testicle out, and severed the artery and vas deferens, together with the ecraseur, the proximal end of the cord falling backwards into the canal and probably into the abdominal cavity; plugged the canal with sterilized gauze kept in by sutures, dusted the wounds and their vicinity with iodoform, and let the horse up.

There was no suspicion from the appearance of the wounds of hæmorrhage taking place. In the evening after the operation the horse took his food well, but his pulse was weaker than normal, and there was a good deal of dark blood dripping from the wound on the normal side. The slight bleeding was easily arrested by inserting a plug of sterilized wool into the scrotum. There was not a trace of blood from the other side. The next morning the horse took his feed, but his pulse was weaker, being difficult to count, but never exceeded 60 in frequency. The temperature was 104° F. I was surprised that there was practically no heat or swelling in the vicinity of the wounds: on the contrary, the sheath and scrotum seemed flabby and coldish. The mucous membranes were not pale, but seemed somewhat cyanosed. The respirations were not accelerated. Soft fæces and urine were freely passed, and there was no evidence of abdominal pain. He took his mash at night. The next morning, the second day after the operation, the horse was blowing hard, and had a very weak pulse and a temperature of 104. The penis hung limp and cold from the sheath, and the extremities were cold. Later he sweated and trembled, and died at 11 p.m., about fifty-six hours after the operation.

*Post-mortem* examination revealed the abdominal cavity almost full of blood. There was nothing about the cords to indicate that hæmorrhage had occurred from them. We came to the conclusion, without positive proof and without looking out for other bleeding vessels, that bleeding occurred from the end of the retained cord which was severed by the ecraseur, after its return to the abdominal cavity, and that bleeding could not show in the inguinal canal owing to its being well plugged. There was no trace of peritonitis. I have always used the ecraseur for retained testicles, and never had bleeding before. This carcase also rapidly became emphysematous throughout the body. The testicle was hard and fibrous, and contained a dermoid cyst.

## MUCO-ENTERITIS FOLLOWED BY MULTIPLE ABSCESSES IN THE SPLEEN, AND PERITONITIS, COMPLICATED WITH HEPATIC CIRRHOSIS AND THROMBOSIS OF THE BRANCHES OF THE PORTAL VEIN IN A RACEHORSE.

By J. A. GILRUTH, D.V.Sc., M.R.C.V.S., F.R.S.E.
*Professor of Veterinary Pathology, University of Melbourne.*

This interesting case was brought under my observation by Mr. E. F. J. Bordeaux, B. ès L., G.M.V.C., Melbourne, in whose care the animal was during the whole course of the illness.

The case is almost unique in many respects, and I was glad to have an opportunity on several occasions to see it, and especially of making a *post-mortem* examination along with Mr. Bordeaux. The onset of the disease, the intermittent colicky symptoms, the progressive weakness, the irregularity of the temperature, and during the later stages of the illness the appearance of jaundice are specially interesting features. From observations of the case I am convinced that death would have occurred at a much earlier date but for the special attention given by Mr. Bordeaux and the careful nursing of the owner.

The following is Mr. Bordeaux's short account of the symptoms and treatment of the case :—

"*Subject.*—Bay thoroughbred horse, aged 4.

"Brought to hospital on November 24, suffering from colic with usual symptoms but no violent struggling. Temperature and pulse normal; contents of rectum slightly mucus-coated, peristalsis slow. I wanted to administer linseed oil but was asked by the owner not to do so as this horse was engaged in a race on December 1, so I gave him arecoline $\frac{3}{4}$ gr. and a ball of nux. vom. and ammon. carb. Enemata were to be given until pain ceased. This horse won the race on December 1, and appeared in perfect health on that day. I advised the owner to put him on a light diet of mashes and green food for a few days, but my instructions were not carried out, the horse being fed and worked as usual.

On December 3, immediately after work, colic symptoms reappeared. The contents of the rectum were mucus-coated; temperature 104° F. Diagnosis, subacute enteritis. I administered Ol. lini. spt. ether nit., and spt. ammon. co. For the next three days the temperature varied from 103° to 101° F. but there was no sign of intestinal pain and the appetite was good; diet consisting of mashes, green food and a little oaten hay.

"On December 6, petechial spots appeared on the membrana nictitans. Slight intestinal pain was exhibited about two hours after each meal (which I considered was probably due to some ulceration of the mucosa of the large colon) and this continued for a week, although the diet consisted only of mashes and green food. Treatment, nux. vom., quinine and gentian. The temperature remained between 102° and 103° F., and the general condition did not alter much until the 13th, when the intestinal pain became acute. The contents of the rectum were thickly coated with mucus and large hæmorrhagic spots appeared on the visible membranes. I administered ol. lini. with spt. ether nit. and spt. ammon. co., after which the the intestinal pain disappeared, and prescribed the following treatment. One pint of cod liver oil and 1 pint of milk morning and evening as a drench, which the horse took without trouble. Nux. vom. and potass. iodid. at mid-day. This treatment was adopted as the diagnosis was then muco-enteritis of the large intestine with ulceration. The appetite was still good, green food only being given. Colicky pains did not reappear.

"On the 18th the temperature was 105° F., pulse 52. The former treatment was continued in addition to which the bowel was flushed twice a day with warm soap and water and ½ oz. of liq. arsenical, given every evening in a handful of damp bran. The temperature varied from 103° to 104° F. for the next few days, the pulse was weaker and more frequent (72), and the horse was getting very thin.

"On the 25th, the membranes had become icteric., and there was extreme weakness, but the appetite was still good. The legs were becoming œdematous, as the horse was now afraid to lie down.

On the 27th, the temperature was 106° F., pulse 80, and general condition very grave. The general condition got worse every day, the temperature remaining high, 106° F. until the 31st, when it suddenly fell and the horse dropped dead at noon."

*Post-mortem* examination was made at the "Knacker's" about four hours after death. The following are the notes. Body emaciated. Subcutaneous tissues yellow and showing areas of hæmorrhage, particularly about the limbs. Peritoneal cavity contains about 2 gallons of yellowish granular fluid with numerous flocculi. Fibrinous adhesions between anterior flexure of floating colon and spleen. Parietal surface of peritoneum congested and studded with small inflammatory papillæ. Omenta swollen congested and œdematous. Stomach, mucosa congested; several tumours due to *Spiroptera megastoma* present (a common condition in Victorian horses). Small intes-

tines and great colon normal. Floating colon at anterior flexure shows several small patches of old submucous hæmorrhage and several circular and oval depressions, livid in colour, being apparently healed ulcerations. Spleen enormously enlarged, weight 37 lb. Enlargement is chiefly due to a large swelling about twice the size of a football, implicating practically the whole of the organ with the exception of a small area at each extremity. On section this swelling is found to consist of a large central mass of semi-fluid, granular, greyish material, surrounded by numerous purulent foci varying in size from a wheat grain to a pea scattered irregularly amongst a dark, almost black, and softened spleen pulp. Liver, enlarged, weight 35 lb.; consistence comparatively soft and friable. On section the majority of the branches of the portal vein are seen to contain long yellow-coloured dense thrombi varying in diameter from that of a thread to that of a pencil. (Unfortunately the vein was not carefully examined before removal so that the condition could not be observed definitely to exist external to the liver.) The thrombi appear at places distinctly adherent to the wall of the vessel, at others lying free. Hepatic lobules swollen and distinct and apparently surrounded by a fine peripheral greenish band which on careful examination gives the organ a reticulated appearance. Kidneys pale and swollen. Pleural cavity contains about a quart of sero-sanguineous effusion. Lungs normal but for one or two emphysematous areas. Pericardium contains about 6 oz. of sero-sanguineous effusion. Underneath the epicardium of the auricles and at the cardiac grooves are large patches of blood extravasation with gelatinous exudate, but otherwise organ normal.

Microscopical examination of smears showed as follows: Peritoneal fluid, spleen pulp and pus, many leucocytes, chiefly polymorphs, with short chains of streptococci and numbers of short cocco-bacilli, gram negative. The central broken-down material in the spleen contained chiefly streptococci and large diplococci; the blood diplococci and cocco-bacilli; the surface of the liver thrombi streptococci, short bacilli, and some large bacilli probably putrefactive; but in the centre of the thrombi no organisms could be demonstrated. Pure cultures of both organisms were secured; the cocco-bacillus exhibiting all the characters of the pasteurella group; the streptococcus having no special characters, being similar to that usually found in purulent affections of the horse.

Microscopical examination of liver sections shows a condition of interlobular cirrhosis of some standing. The new fibrous tissue

implicates chiefly Glisson's capsule, but is found often invading the lobules, causing atrophy and distorsion of the liver cells. New bile ducts (so termed) are frequent. The bile ducts and branches of the portal vein are implicated in the inflammatory proliferative process and it is most probable that the thrombosed condition of the portal vein branches, observed microscopically, is due to extension of this through the vessel wall to the intima. The thrombus in each small portal branch is almost invariably attached to the intima but at only one area of the periphery, the remainder being free. In the space between the free surface of the thrombus and the intima of the vessel normal blood cells are usually found indicating that some circulation was being maintained during life throughout the liver. Catarrh of the bile ducts is absent. The hepatic cells are loaded with greenish pigment as is so often the case in hepatic cirrhosis of the horse.

The thrombi show no evidence of streptococci or other organisms and none can be detected in the liver substance proper.

*Conclusions.*—The pathology of the case is extremely interesting. The *post-mortem* picture was complicated, and taken together with the clinical history of the case, the correct reading would seem to be as follows:—

The animal was primarily from the clinical point of view affected with a sub-acute muco-enteritis of the colon; the cause being probably the pasteurella of influenza (a common equine affection in Melbourne), Streptococcic invasion followed as so often happens and extended direct by contiguity, to the spleen and the peritoneum. So far as these regions are concerned the explanation suffices, but the liver lesions remain unaccounted for. At first one is tempted to conclude that the hepatic thrombi and cirrhosis were due to further extension of the streptococcic infection, but the absence of streptococci in the liver substance and especially within the thrombi negative this. It is more probable the hepatic lesions were independent, due to some toxic agent in the fodder to which the thrombus was secondary. We are aware that the plants of the *Senecio* family will gradually produce a definite hepatic cirrhosis in horses, cattle and sheep (as was first experimentally demonstrated by me—see Department of Agriculture, New Zealand Reports, 1902), and subsequently by others in Canada, South Africa, &c. In this particular case no evidence of *Senecio*-contaminated fodder was forthcoming, but seeing that *Senecio jacobæa* (Ragwort) is fairly common in certain agricultural districts of Victoria, and that this horse had been stable-fed for two years on market-purchased fodder, the possibilities of such a cause having been in

operation must not be overlooked. Further, it has not been satisfactorily shown that bacterial infection can produce such a general cirrhosis as presented by this case. On the contrary, my experience is that a horse may present no symptoms whatever of illness, although the liver be in an advanced stage of cirrhosis, provided the alimentary canal remains normal, but that the slightest digestive derangement is followed by severe, and generally fatal results, possibly due to the inability of the liver to cope with and neutralize the intestinal toxins.

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## Canine Clinical Notes.

### TORSION OF THE STOMACH IN A DOG.

By JOHN L. PERRY, M.R.C.V.S.

*Cardiff.*

A VALUABLE bloodhound was brought to me at 2 a.m. on August 26, the owner's message being that he thought the dog had "picked up poison." The history was that the dog had been taken out for exercise the previous evening, and returned home apparently healthy. When fed with melox and paunch, cut up, at 11 p.m., he was noticed to eat a little, then walk round his kennel and return to his food, again eat a little, then repeat the performance. Soon afterwards he commenced to paw his bedding about and moan frequently. This continuing for some time, the owner administered an emetic of salt and water, which was swallowed all right. Soon afterwards there were constant attempts to vomit, dribbling of saliva, swelling of abdomen (tympany), and noisy eructations of gas.

All these were in evidence when brought to me at 2 a.m. the following morning, the dog being extremely restless and in acute pain.

The abdomen was quite hard and tense, and manipulation increased the pain. I gave him 1¾ gr. of morphia subcutaneously and a full dose of sod. hyposulph. *per os*. In half an hour's time the tympany was considerably less and the pain diminished. Although neither vomition or defæcation had taken place, the soporific effects of the morphia were pronounced, and advantage was thus taken to secure the dog on the operating table and pass the probang. Some obstruction was encountered at the stomach end of the œsophagus, but no opinion could be formed as to its nature; it could not be complete blocking, as swallowing could take place, although it increased the retching.

The dog remained under the influence of the morphia for four hours. After that small doses of sod. hyposulph. and chlorodyne were administered, the patient lying fairly comfortable, except for an occasional twinge of pain, until 10 a.m., when the tympany and pain got worse. A fine trochar was then inserted into the stomach on the left side, and some gases evacuated followed by a little fluid. This gave only temporary relief, and the dog died at mid-day.

A *post-mortem* examination was immediately made, and revealed the stomach twisted on its superior attachments—from left to right—a half turn. The whole organ was very dark, almost claret colour, and engorged with blood, owing to vascular compression. The contents were about a pint in capacity—equal parts liquid and solid; amongst the latter were found pieces of paunch. After adjusting the organ to its normal position, the hole made by the trochar was sought and found on its right side.

I cannot find any mention of torsion of the stomach in any English volume, but I am told that Cadiac, in his "Encyclopædia Vétérinaire," says the condition is not uncommon, and that he saw three cases in one year. He says the anatomy of the organ permits free torsion from left to right, especially when the organ is empty.

Of course, no treatment can be of any avail in such cases, excepting some surgical interference, such as a laparotomy performed in the early stages.

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## AN INTERESTING CASE OF NETTLE-RASH IN A DOG.

By E. RAITSILS.

When walking in the street a six months old pointer suddenly and unexpectedly developed a severe œdematous swelling of the whole head, and of the œsophagus, together with the upper part of the under surface of the neck. The swelling was diffuse, not very hot, not sensible to pressure, and felt doughy. In addition, on the skin of the sides of the neck and anterior extremities there were round elevations of the skin, from the size of a lentil to a horse bean. After a dose of salts and friction with spirituous lotion the swellings had completely disappeared on the following day. As the dog consumed different kinds of vegetables daily, it appears not unlikely that the rash was due to some plant material. The author has noticed a similar case in a fox-terrier that had an attack of nettle-rash regularly after eating strawberries or chocolate.

(*Deutsche Tierärztliche Wochenschrift.*)

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# Abstract.

## THE RELATION OF PURE SCIENCE TO PRACTICE, WITH SPECIAL REFERENCE TO THE PHENOMENA OF PARASITISM AND INHERITANCE.[1]

By H. W. MARETT TIMS, M.A., M.D., F.L.S.

*Professor of Biology, Royal Veterinary College, London.*

In my student days it was customary to devote a considerable portion of an introductory address to the delivery of a homily upon the correct conduct of youth. In Shakespeare's words we were bidden—

> To live and study here three years.
> But there are other strict observances;
> As, not to see a woman in that term;
> And, one day in the week to touch no food;
> And, but one meal on every day besides;
> And then, to sleep but three hours in the night,
> And not be seen to wink of all the day.

It is not my intention, however, to take up your time in this fashion; were I to attempt to do so, your answer to such would doubtless be the same as that of Birón to the exhortion just quoted—namely :—

> O, these are barren tasks, too hard to keep;
> Not to see ladies—study—fast—not sleep.

Nevertheless, without unlocking a stream of moral platitudes, I would wish, on behalf of my colleagues and myself, to offer sincere congratulation to the prize winners, may they "go on and prosper," and to those now entering upon their college career we extend a sincere welcome. I hope you will soon feel at home, and that you will from the first take a real interest in all the activities of your College life, its sport, and its societies.

The true value of a collegiate life is derived from the judicious combination of the intellectual and social sides. The one fits you for your professional career, the other (and it is of scarcely less importance) teaches a man "to aid and bear a part in all actions and occasions." It should, moreover, be the period of life for making friendships, for "Whosoever is delighted in solitude, is either a wild beast or a god," to quote the words of Aristotle. I *know* you are not wild beasts. I *presume* you are not gods. Friendships are, therefore, very necessary, for "if a man have not a friend he may quit the stage."

I would, therefore, urge upon all who are now joining to take an active, but not a too engrossing, share in the college games. Cultivate *esprit de corps*. The success of the collegiate life in the older universities is largely due to this spirit. It is what we of the London Colleges, more particularly those colleges constituting the University of London, and among the number of which I hope this College will very shortly be included, require more and more, and which we ought to do all in our power to foster. A healthy spirit is just as necessary

[1] Introductory address at the opening of the session of the Royal Veterinary College, London.

for the life of an institution as of an individual. It is, however, to other subjects that I wish to draw your attention for a short time to-day.

Not being a member of your profession, it would be an impertinence on my part were I to attempt to address you upon a subject immediately concerned with veterinary practice or politics. I have therefore selected a more general topic, and one upon which I feel myself somewhat more competent to speak, the relation of so-called "pure science" to practice with special reference to the phenomena of parasitism and inheritance, and incidentally the place and importance of biology in a medical curriculum. I may say in passing that in using the terms "medicine" and "medical" I refer to both human and veterinary, for the problems in both cases are similar, and I believe the educational aims to be identical.

It is often asked by the student and by the "practical" man, What is the use of biology in the medical curriculum? Why should the student be required to waste time over the anatomy and physiology of animals, for which he will never be required to prescribe, or at whose confinements he will never be called in to assist? Such questions are not altogether unnatural from a student in his first year, since he cannot be expected to realize the full import of the subject. The more serious objector is the fully-qualified man, the post-graduate, whose proud boast is that he is nothing if not practical, and who is ever insisting on the fact that medicine is a practical subject. It is to such an one that I would address my remarks, and attempt to show that, while not underrating in the very least the value of the practical applications of science, nevertheless it is to the pure biologist (using the term in its widest sense) that nearly all the more important advances in biological knowledge are due, to prove in fact what the President of the British Association so recently said at Sheffield, that the modern Universities have to prove "that abstract thought is not antagonistic to practical work, or scientific research to the labour of the factory or foundry," and, may I add, "or practical medicine." "The one and the other can harmoniously co-operate in the advance of knowledge and the progress of civilization."

The pure science man works at his subject with the sole object of acquiring and extending knowledge, of investigating the laws of Nature without any particular thought of practical applications. This is beyond doubt the highest aim. Unfortunately, among such there is sometimes a tendency to belittle the practical side; indeed, some extremists appear almost to go so far as to think that if a subject can be shown to have an economic or utilitarian aspect it ceases to be, if not scientific, at least to be of less interest. In my opinion such views are unfortunate, and such men lay themselves open to the stigma of being regarded as visionaries, or even "cranks," with but little knowledge of the world. On the other hand, the purely practical man is often a man of equally limited outlook, too apt to gauge every scientific fact by the sole standard of utility, and the utility is but too often estimated by the monetary value derivable from the application of the scientific facts. For, if we analyse this modern cult of the practical, I think one is bound to admit that the main underlying considerations are but too often mercenary.

While fully realizing that the majority of students are destined

to become practitioners, earning their livelihood by the practical application of scientific facts, I am afraid there is a danger of bending the knee too much and too often to the Baal of practical utility. We require, I think, to realize more fully than is often done that the "pure scientist" and the "practical man" are both equally necessary, that one is the corollary of the other, and in this faith I would urge that medical education should be as broad as possible, and not regulated entirely by motives of immediate utility. The student, while his mind is in a receptive state, untrammelled by the cares of income-earning, should be taught the more theoretical aspects of science, upon which his future practice should be based; his mental outlook will thereby be broadened, and I venture to think that the student who has been trained in general principles and scientific methods will, in nine cases out of ten, turn out to be the better practitioner.

Faraday, the results of whose researches have had such far-reaching effects, was one day asked by a lady in a somewhat depreciatory tone what was the use of his researches? His simple but pregnant reply was, "Madam, what is the use of a baby?" I think it may fairly be stated, at any rate of the biological sciences, that all the most valuable practical results have been the outcome of researches which in the first instance, and taken by themselves, appeared to have no value beyond that of scientific interest.

Who, for example, would have imagined at the time that the simple experiment of Van Helmont would prove to be the starting point of such important and far-reaching results? Van Helmont planted a willow tree of known weight in a tub of dry soil also of known weight. After a period of five years both were again weighed, and from the resulting differences certain deductions were drawn as to the transformation of water into the material of the tree. This apparently useless experiment in reality was a basis upon which, as Mr. A. D. Hall has recently pointed out, the super-structure of modern and rapidly extending science of agriculture has been erected.

Again, turn to the academic researches of Pasteur upon the growth and nutrition of the yeast plant, his work in connection with fermentation, and perhaps more than anything else his researches, commenced in 1865, into the causation and prevention of silkworm disease, and note how they paved the way for the dominating germ theory of disease. To quote the words of the distinguished American Protozoologist Calkins [2], it is of no importance that the characteristic black spots of pébrine were not recognized by Pasteur "as the spores of a protozoon, but the important results which followed their discovery, and which led to increased length of human life and to the mitigation of human and of animal suffering throughout the civilized world, would make an increasingly substantial monument to the patience, courage, and virility of this man of pure science, who, by the apotheosis of scientific method, proved these unknown corpuscles to be the cause of this silkworm disease."

The controversies over the cell-theory and the bitter controversy over the theory of spontaneous generation brought the knowledge of the protozoa, both parasitic and free-living, prominently before the biological world, but not till nearly a quarter of a century later did the protozoal origin of disease find any acceptance, and it is only within the past few years that these ideas have become unshakably

established. Though the researches of Pasteur did so much to stimulate the study of micro-organisms, members of the two great groups of pathogenic protozoa, the hæmoflagellates (to which the trypanosomes belong) and the sporozoa were known before Pasteur's work on the silk-worm disease. The first trypanosome appears to have been discovered by Valentin in 1841 in the blood of the trout. Very shortly afterwards Remak and Berg noted their presence in other fishes; then followed the discovery of these organisms in the blood of frogs, birds and rats. The first trypanosomes met with in disease was by Griffith Evans in 1880 who found them in horses suffering from Surra in India. Then came Schaudinn's classical work on the *Trypanosoma noctuæ* of the little owl, and Bruce's brilliant researches in 1895 when he discovered the parasite of nagana, the tsetse-fly disease, in the blood of infected horses and cattle, and the important part played by the fly in transmitting the parasite.

With the more recent work on sleeping-sickness and Malta fever you are all familiar.

So too, with the sporozoa, undoubted Gregarines were noted in invertebrate animals before the close of the eighteenth century, and as early as 1839 Hake published investigations upon the spores of the coccidium of the rabbit. In 1841 Johannes Müller described many new forms in fishes. These led on to other researches which culminated in the discovery in 1880 of the malaria parasite by Laveran.

Koch regards the discoveries of Bruce and of Laveran, together with that of the parasite of Texas-fever in cattle in 1893 by Smith and Kilborne, as the three great landmarks in the history of our knowledge of the pathogenic protozoa, each of which opened up new and wide fields of fresh investigation, until now as Professor Minchin [7] has said "So great is the interest which these parasites excite at the present time, on account of their pathogenic properties in man and beast, that now scarcely a month passes without the publication of some discovery relating to them, and the study of the protozoa bids fair to assume in the near future a position of importance scarcely secondary to that held by the science of bacteriology."

Thus we see that the early discovery of these important organisms was due to the labours of men of pure science, who found them in the lower vertebrates and in some invertebrates, and that they, unimportant as they appeared to be in themselves, formed the basis of the later work which has been of such incalculable benefit. Indeed, it is safe to say that bacteriology and the whole principle of antisepsis and asepsis as well as of protozoology take root in the classical researches of such men as Pasteur. The discovery of the causation of these protozoal diseases has led on to their prevention and treatment. Nuttall's discovery of a cure for piroplasmosis is of the utmost importance and the reduction of animal mortality likely to ensue will lead to the saving of thousands of pounds annually. Is it too much to expect that the active researches now being prosecuted all over the world into the causation of cancer, to which the cytological researches of Farmer and Moore gave such an impetus, may before long lead to similar practical results?

Turning now to the metazoan parasites. The more systematic study of their effects upon the blood of the host has yet hardly been

entered upon or its importance so generally appreciated. The life-histories of some of the commoner cestode and nematode worms have been known to us for several years, and for a great deal of this knowledge we are indebted to our own countryman Cobbold, and a former distinguished lecturer in parasitology at this College, but there is yet much to be done. The brilliant and patient researches of Loos [7] extending over many years and from which he came so near to sacrificing his own life have shown conclusively that the larvæ of *Ankylostomum duodenale* can penetrate directly through the human skin and that of puppies when brought into contact with water containing the larvæ. In Neumann's "Animal Parasites" [10] it is stated that "Humidity has at all times been considered as one of the most predisposing causes to the invasion of the economy by worms. Rainy years are marked by the extension of various forms of helminthiasis in animals which graze; and these affections are more marked among animals that frequent inundated pastures and swampy places, and the borders of lakes and ponds." This is explained by the ova being preserved in water and being taken in when drinking. May there not be a possibility, at any rate in some cases, of a direct infection through the skin such as Loos has shown to be the case in the miner's worm. If such a possibility should turn out to be correct, then mere attention to the drinking water would be of no avail, the animals should not be allowed to frequent "swampy places or the borders of lakes and ponds." It seems to me that such a question would be worth while investigating.

It may be said that the presence of intestinal worms is on the whole of comparatively little moment since so many animals are infected with so little proportionate disturbance of the economy. Krabbe states that 67 per cent. of all dogs have intestinal entozoa. This objection may be true to some extent as regards certain entozoa being the direct and immediate cause of serious symptoms, but there is no doubt that they may act as the indirect and predisposing causes of much more serious affections. Their mode of attachment to the intestinal wall causes a lesion through which bacteria or other organisms may gain admission to the blood and lymph. This point has been insisted upon by Dr. Shipley [9] who considers that many cases of appendicitis in the human subject associated with the presence of parasitic worms in the intestines and in the appendix itself, are to be explained in this manner. Further, evidence in the same direction seems to be forthcoming in the findings of the commission into the causation of grouse diseases.

But I do not think we are even here at the root of the matter; there is, I believe, in many cases something more. The parasite is not in all cases the mere mechanical agent so to speak, preparing the way for the entrance of other organisms to perform their deadly work. The convulsions, the spasmodic movements, and all the other nervous symptoms to which infected animals are liable are usually ascribed to irritation of the nerve ending, that is to say to mechanical causes. There is reason to believe that the parasite in itself is injurious.

I wish to draw your attention to some recent interesting and remarkable facts concerning parasitism in the crustacea. It is well known that certain crabs are liable to be infected by another parasitic crustacean. The male crab-host is easily recognizable from the

female by certain well-marked differences in the secondary sexual characters, differences in the appendages, in the breadth of the tail, and in other respects. The parasite commences life as a free-swimming form but ultimately becomes attached to the abdominal appendages of its host and there undergoes degeneration, becoming in time converted into a globular mass, without definite organs, and from which spring long processes penetrating into the tissues and organs of the host. The effects of this parasite upon its *male* crustacean host are most remarkable; the secondary sexual characters are transformed into those of the female, so that the infected males have been constantly mistaken for females.

In addition to this Giard [5], Geoffrey Smith [11], and Potts [8], have shown that the effect of the parasite is to cause dwindling and ultimately an entire disappearance of the genital glands, and on account of this effect Giard gave the name "Castration parasitaire." Infected females show very little modification of the external characters, but the ovary is reduced or obliterated. The still more remarkable observation has been made that the completely modified males may occasionally recover from the parasitic infection and regenerate the genital gland, in which case they become perfect hermaphrodites, the gonads producing both ova and spermatozoa. Comparable phenomena are now known to occur in nearly all classes of invertebrate animals, though the alteration of the secondary sex characters is not so marked a feature as in the crustacea.

In Mozambique there is, according to Turner [12], a very prevalent idea that impotence and sterility accompany bilharziosis but I know of no definite proof of the interference with fertility among vertebrate animals due to parasitic infection. Nevertheless, I think it is a point worth bearing in mind as it might be of some practical importance in relation to the value of infected animals for breeding purposes.

What these researches do show is that the parasite has a very marked effect upon the metabolism of the host. How that effect is brought about, whether through the action of a secretion into the body of the host, or whether by some interference with the normal hormones of the host, as has been suggested, it is as yet quite impossible to say. I would suggest that a useful line of work in the first instance would be to make an extract of the crustacean parasite and inject it into normal uninfected males and see whether effects similar to those caused by the parasite itself can be produced, and thus to determine the point as to whether or not the effects are caused by a secretion from the parasite.

I would note in passing that the parasites known at present to cause deterioration of the genital glands are those found in the body cavity or in the blood spaces and not the gastro-intestinal parasites, but that these latter have in some cases a marked effect upon the blood of the host has been amply demonstrated. A good deal of research has been devoted to the changes produced in the human blood by various endo- and ecto-parasites, some of the more important results of which I may briefly mention.

Of the Cestoda, *Tænia solium* and *T. saginata* have but little effect and that of a transitory nature, the effect being limited to the Eosinophiles, but in the Cysticercus stage there is marked eosinophilia. On

the other hand the results of infection by *Dibothriocephalus latus* are of a much more serious nature. In the blood of Tænia-infected persons the leucocytes, as I have just said, only are affected, whereas in Dibothriocephalus infection the effect is almost entirely confined to the red cells, in many cases giving rise to so profound an anæmia that it is only distinguishable from true pernicious anæmia by the fact that the patient recovers on expulsion of the worm. The character of the blood in these cases is "a marked oligocythæmia, a high colour index, the presence of nucleated red cells of the megaloblastic type, the appearance of deformed red poikilocytes and cells showing polychromatophilic staining" [4].

Hydatid disease due to the cysts of echinococcus gives rise to a general and serious leucocytosis.

Of the nematodes, cases of infection by the common *Ascaris lumbricoides* are reported in which, in addition to an eosinophilia, the red blood-cells have been reduced to half the normal number.

The very marked anæmia of Egyptian chlorosis due to infection by *strongyloides intestinalis* is well known to everyone.

It is not necessary to multiply examples, but the point upon which I wish to insist is that when one takes into account all the facts of parasitic infection, we are forced to the conclusion that the parasite generates some poison which affects the host to a greater or less extent, and that the leucocytosis is probably a protective effort on the part of the host. The anæmia is to be regarded as a result of infection and not merely a predisposing cause, as stated in some of the text-books; and, further, that this anæmia is due to some toxic influence from the parasite. I suppose that it is possible that immunity to those effects can be established, and for that reason the frequent infection of dogs does not give rise to more serious symptoms than it does, though, as we have seen, the cestodes even in man give rise to but slight changes in the blood.

Is it not probable that not only the anæmia, but all the other symptoms of helminthiasis—the convulsions, epilepsy, vomiting, &c.—are all due to this toxic affection?

There is a sentence in Neumann's book which seems to me to point out a line for further researches. He writes: "There are symptomatic manifestations that properly belong to each kind of domesticated animal and to each species of parasite." May this not be indicative of a different toxin (I use the word for want of a better in our present ignorance) for each parasite, or of differences in the chemistry of the blood of the different kinds of host, or of both?

It seems to me that there lies here before us a wide field for research, for the further application of the opsonin theory, and possibly paving the way for an antitoxin treatment of helminthiasis.

Let me turn now to a subject of even greater importance.

Of all the advances in biological science of recent years, none are of wider interest or of greater practical importance to the breeder, and one maysay to the human race, than the discovery of certain principles of heredity by Gregor Johann Mendel. This great man, born on July 22, 1822, at Heinzendorf, of Austro-Silesian parents, entered as a novice the Augustine Convent at Altbrünn, and afterwards became a teacher of natural science at the Realschule in Brünn. It was during this latter period (1853-1868) that he devoted much of his time to the

cross-breeding of a large variety of plants. Upon his appointment as Abbot of his monastery in 1868, his scientific activities were considerably curtailed by the duties associated with his new office; nevertheless, the work of the preceding fifteen years had been sufficiently great, and his records so careful and exact as to have formed a very solid foundation upon which a vast superstructure is being daily raised by workers in all countries.

Mendel's first experiments in hybridization were made with the eating pea—*Pisum sativum*—and his results were published in the *Proceedings of the Natural History Society of Brünn* in 1866. For over thirty years his work remained unknown, until in 1899 the main facts were rediscovered by de Vries, Tschermak, and Correns; and in our country they have been brought prominently before the world by Bateson, who, with his pupils Punnett and Doncaster, has added so greatly to our knowledge of heredity. Before drawing attention to the great practical application of Mendel's work, I may briefly outline the principles, taking for illustration one of his own experiments.

He selected plants with pairs of characters such as tallness and shortness, and made crosses between the tall and short varieties, selecting only those which he had proved to breed "true." Tall plants alone resulted from the first crossing (F 1). The character of tallness he called Dominant, and that of shortness he called Recessive. The seeds from these tall forms were collected and sown again the next year, and in the resulting second generation (F 2) some plants were tall and others short in the nearly accurate proportions, 3 to 1. If the seeds of the dwarf forms were then planted they gave rise to nothing but dwarf forms for however many generations they were carried on—*i.e.*, they bred true. But if the seeds of the tall forms were planted, it was found that they again gave rise to tall forms, but by growing them through further generations, he showed that they were not all of the same nature—some were pure tall, others impure tall, in the proportions of 1 to 2—that is, that the pure tall now breed true like the recessives of the previous generation, whereas the impure dominants (tall) again yield dominants and recessives in the constant proportions of 3 to 1. We thus see that a tall form may be a pure dominant, and will therefore breed true, or it may be an impure dominant—*i.e.*, though it appears as a dominant tail form, it really carries the recessive character of shortness within, though latent, as proved by the fact that some of the descendants of the next generation will exhibit that dwarf character.

Large numbers of characters have been dealt with in the same way both in plants and animals. The coloured coat of mice and rabbits is dominant over the albino coat. The short fur of rabbits is dominant to the long "angora" coat; the "rose" comb of Wyandotte fowls is dominant to the "single" comb of Leghorns and Andalusians.

Professor T. B. Wood has also shown, by crossing Dorset horn sheep with the Suffolk hornless breed, that horns are dominant in males and recessive in females [17].

Cases are, however, met with in which the hybrid of the first generation is intermediate in character between the two parents, as is shown by crossing a lax-eared with a dense-eared wheat. But when the seeds of the hybrid are sown the next generation (F 2)

they give one quarter dense, one quarter lax, and two quarters intermediate, the last again behaving in the same way in the next generation. The dense and the lax thus reappear after skipping a generation, and then continue to breed true.

In an address of this nature it is impossible to go into the various explanations of the results and of the various theoretical deductions as to gametic segregation, gametic coupling, &c.; but they are all adequately dealt with in books devoted to Mendelism. I wish now rather to draw your attention to experiments on the same lines in respect of characters of marked practical importance.

Professor Biffen, of Cambridge, finds that there is a variety of wheat which is rust-proof, a small-eared variety without any good qualities beyond its immunity to rust. Another variety is exceedingly susceptible to rust, but formed long ears. These two varieties have been crossed, and Biffen finds that immunity to rust acts as a recessive character. By definitely selecting the crossings through several generations it is found possible to get a wheat "which will bake as well as the best Canadian, and, since they will be free from disease, crops better than any varieties at present on the market" [13]. To quote again from a paper by Professor Wood and Punnett, this "possibility of transferring immunity to disease from one variety to another is an achievement of the greatest scientific and practical importance. It seems to offer the most hopeful method of checking disease yet suggested. If immune strains of any species of plant or animal can be found, no matter how useless they may be from other points of view, there seems to be no reason why their immunity should not be transferred by crossing to our present valuable varieties which are being ravaged by disease. In this way it may be found possible to produce high-class horses and cattle immune to the diseases which are the scourge of the South African farmers, sheep which will be proof against anthrax, black quarter, and other diseases that attack sheep at home, and pigs immune to swine fever."

It may be that some may regard these possibilities as Utopian, and not justified merely by what has been found possible in wheat. Is there any evidence that disease in animals behaves as a Mendelian character? From the nature of the case the evidence in animals is as yet much more scanty than in plants, but it is quite sufficient to raise most sanguine hopes.

Some very remarkable pathological examples of Mendelian heredity in the human being are the cases of brachydactyly investigated by Farabee in America and Drinkwater [3] in Edinburgh. This condition, affecting hands and feet, and associated with short stature, is a dominant character and behaves in a simple Mendelian way. Without going into details, it is possible from a study of these cases to confidently prophesy that no unaffected member of the family will transmit the disease to his or her children, but that it will most certainly be transmitted by the affected members, and by them only. Congenital cataract is another condition which seems to behave as a simple Mendelian dominant to the normal [8].

These are comparatively simple cases, but there are those of sex-limited diseases where the disease is present generally, if not always, in members of one or other sex only—for example, males; in such cases the females, unaffected themselves, may, and usually do,

transmit it to their sons, but not to their daughters. This is the case with some forms of hereditary night-blindness; the males only are affected, with descent through the normal-sighted females [8].

Several other diseases are under investigation—hereditary chorea, diabetes insipidus, deaf-mutism, hæmophylia, and many diseases of the eye and nervous system [9].

It is getting now possible to predict with some degree of accuracy which of the offspring, male or female, of an affected person will in turn be affected. Beyond prophecy and advice, little can be done with human beings; "Man will aye gae his ain gait," and "Wilful woman will have her way." But in the case of animals this is quite different.

May we not see here opening up a field of vast importance to the breeder and to the veterinary surgeon? One more point before I close. I would like to draw your attention to a very suggestive paper by Major F. Eassie, D.S.O., of the Army Remount Department [3], published in the *Scientific Proceedings of the Royal Dublin Society* for March last, on "Some Variations in the Skeleton of the Domestic Horse, and their Significance." The authors gives proofs of deterioration in the domestic horse, as compared with the wild horse.

Though the evidence furnished is not sufficient to state that the points in a horse dealt with in the paper do behave as Mendelian characters, the paper seems to me to be extremely suggestive of the lines upon which breeders might work in the selection of their animals. As the breeding of horses and the improvement of the breed are now very much to the front, I would commend this paper to your notice.

Time will not permit me to go further into these interesting and important subjects. I hope I have said sufficient to prove the thesis with which I started—that all these matters, whether in regard to parasitism or heredity, are of immense practical value, and that they all had their origin in the apparently useless work of the pure and unpractical scientist. We see here an answer to Faraday's question, What is the use of a baby?

Further, I hope I have successfully demonstrated the value of a knowledge of biology to the student, and its importance as a subject in the veterinary and medical curricula. The shafts so constantly aimed at biology should as a rule be aimed not at the subject but at the syllabuses which educational authorities lay down. Taught, as it should be, with due regard to general principles, I hope the day when biology is banished from our medical colleges and relegated to the position of a school subject may never come. It would then be taught by untrained persons whose sympathies might be in entirely other lines, possibly not even scientific, and without any regard to its future application by the student.

It seems to me to be becoming more and more evident that the centre of gravity of medical education is shifting from the pathological to the biological laboratory, and hence rather than crushing the subject it will have in the future to be more and more fostered. The great importance of biology has been recognized in the University of Cambridge by the foundation within the past few years of two chairs of biology: the Quick Professorship held by Professor Nuttall and devoted more particularly to the subjects of protozoology and parasito-

logy; the other chair now occupied by Professor Punnett, and previously by Professor Bateson, to the study and investigation of all problems connected with heredity.

I am aware that we are all open to the charge of believing that our own particular subject is of all others *the* most important; but I do seriously think that the importance of biology in the medical curriculum is not sufficiently appreciated. In addition to the general elements taught to first year's students, I think a second and more applied course should be taken by the more senior students concurrently with their pathology, dealing not only with parasitism and heredity, but with the animals which are so important in the transference of disease—leaches, flies, ticks, mosquitoes—and now even carcinology is coming to be recognized as of importance.

I am of opinion that the biological departments of our medical colleges should be given functions, wider and more important than the teaching of first year students in the elements of biology, as a sort of introduction to anatomy and physiology and to fill up their time until they have passed in chemistry and physics. Biologists ought to take an important share in the teaching of our senior students and post-graduates. I am referring not to this college or to the veterinary curriculum only, but to all medical colleges, all medical education. And above all I would like to see them becoming centres for research. Research I regard as a most important factor, stimulating both teacher and taught. Where no research is carried on, both teaching and learning are apt to become mechanical, lifeless, and uninteresting.

Your profession, extending as it does throughout the world, has its focus in this metropolitan College. There is no other place where such subjects as the *phenomena* of parasitism (not merely the collecting, identification, and cataloguing of specimens) and the hereditary transmission of disease, and I would add also, of desirable qualities in animals—for this is of equal importance—could be better studied, owing to the vast stores of available material.

May I conclude with a practical suggestion? It is that there should be established in this College a central bureau for the collecting of data, supplied by the members of your profession who are in practice and in touch with the material, and who have such ample opportunities for helping. The collecting of such data would give them an active interest in the work, keep them in touch with their College, and would add greatly to our knowledge.

The ever-recurring objection, want of money, will be at once raised. Would it not be possible to form an association of former students, each member subscribing a guinea or even half a guinea a year, to found a post-graduate research studentship to investigate some of these important and pressing questions? As soon as some practical results were forthcoming I have sufficient faith that some of our more wealthy men, interested in horse and cattle-breeding, would come forward and help. As men of affairs they naturally will not put money in a slot from which nothing is to be extracted.

Finally, I would commend this suggestion not only to the Governors of this College but also to the notice of the Royal College of Veterinary Surgeons. They could do no work more useful to the profession and to the community at large than to establish a department for collecting data, and working out all questions relating to heredity in animals.

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## BACHELOR OF VETERINARY SCIENCE DEGREE, UNIVERSITY OF MELBOURNE, AUSTRALIA.

THE first examination for this degree was conducted by the University of Melbourne during August 1, 2, 4, and 5.

The subjects were advanced surgery, pathology, and bacteriology, the contagious diseases, parasitology, State and municipal hygiene, and plant pathology. The written examinations occupied ten hours, the practical examination in pathology, bacteriology, and parasitology, four hours, and the oral examinations ten hours. The Board of Examiners consisted of J. A. Gilruth, D.V.Sc., M.R.C.V.S., F.R.S.E.; A. J. Ewart, D.Sc. (Oxon., Lond., &c.), Ph.D.; W. Stapley, M.D., M.R.C.V.S.; G. Sweet, D.Sc.; J. White, D.Sc.; S. S. Cameron, D.V.Sc., M.R.C.V.S.; and Sydney Dodd, D.V.Sc., F.R.C.V.S. (of Queensland).

The following gentlemen (who are graduates of the old Melbourne Veterinary College and had attended a special post-graduate course), passed the examination and will be admitted to the degree: Messrs. C. C. Cherry, E. A. Kendall, E. A. Le Souef, W. B. Lloyd Green, N. Macdonald, W. A. N. Robertson, E. A. Weston, and A. B. White.

## THE GLASGOW VETERINARY COLLEGE.

(INCORPORATED.)

THE Forty-seventh Session of this Institution commenced on Wednesday, September 28. The Board of Governors have been fortunate in arranging for William Freeman Barrett, Esq., F.R.C.V.S., President of the Royal College of Veterinary Surgeons, London, to deliver the Introductory Lecture, which is to be given on Friday, October 21.

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# Translations.

## A CASE OF DISEASE OF THE CUTIS AND SUB-CUTIS IN THE HORSE.[1]

BY CHIEF VETERINARY SURGEON ERNST KRUGER.

A HORSE treated in 1908 for an unknown skin disease continued in hospital during 1909, and was finally killed as incurable. External treatment with sublimate and bacillol solution dermatol in substance and in ointment form, together with extremely careful skin hygiene and dietetic supervision, only caused a slight improvement in the malady and of bodily condition. The internal administration of potassium iodide, formic acid, and so on, did not favourably influence the complaint. Microscopic and bacteriological examination gave no information as to the cause or character of the complaint. It was noteworthy that the horse showed lameness first on one foot and then on the other, and sometimes on two or three at the same time. The lameness mostly occurred suddenly. Examination showed an acute painful inflammation of the laminæ; the horn of the white line as well as that bounding the sole and wall was sodden and yellowish coloured. After poultices, made with vinegar, applied to the feet, the lameness soon declined. By extension of the skin inflammation to the laminæ, this laminitis was explainable. Towards the end of the year 1908 diagnostic tuberculin inoculations were made; there was a rise of temperature very similar to the typical tuberculin reaction in tuberculous men, and together with the other symptoms shown, a suspicion of tuberculosis began to be entertained. During the last weeks of the illness of the horse, and when at rest, the respirations were 16 to 18 and deep, whilst in the area of the under third of the left lung a muffled sound could be heard. No nasal discharge was present. Examination of the accessible lymph glands showed nothing abnormal with the exception that the gland at the upper half of the œsophagus on the left side could not be felt. Very soon after the tuberculin inoculation there was a drying up and healing of the skin affection, as well as an improvement of the appetite and general condition. The dry scabs fell off in great coherent sheets, and the coat became glistening again with the improvement in appetite. The improvement lasted four or five weeks, but very soon after the skin

[1] From the clinic of the Imperial Military Farriery School at Berlin, Annual Report, 1909.

affection, as well as the general bad symptoms, set in with renewed severity, on which account the owner, after nine months further treatment, decided to have the horse slaughtered.

*Post mortem* showed a considerable number of nodules from the size of a lentil to a hen's egg, in the spleen, liver, in the serosa of the stomach and intestine, on the peritoneum and pleuræ; the portal, the mesenteric and the inguinal glands were swollen as large as one's fist. The microscopical and bacteriological examination in our laboratory showed that the changes were due to tuberculosis. To find out further whether the illness was due to the *Bacillus typus humanus* or *bovinus*, the whole of the material from this patient was sent for examination to the institute for infectious diseases. Just before publication of this report the announcement came to hand that examination had shown the complaint to be due to the *typus humanus* organism.

(*Zeitschrift für Veterinärkunde.*)

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## A CASE OF INTERNAL SUPPURATIVE OTITIS IN A DOG.

By M. MATHARNAN.

*Veterinary-Surgeon to the Remount Depôt at Caens.*

A FOX-TERRIER, aged 14 months, the property of Captain X., showed auricular catarrh of the right ear since May, 1909. Since the appearance of the malady the animal had been entrusted to me to carry out appropriate treatment. All the series of drugs to combat this complaint had been tried without result. Daily lavages of the affected region with warm boracic solution, followed by oxide of zinc ointment, 1 in 8, to the interior of the ear, iodized glycerine, dermatol, salicylated vaseline, employed successively, yielded no good result. In spite of this treatment the suppuration of the ear never dried up; it was never very abundant, but finally it became of a fœtid odour. The general state of the subject was always satisfactory.

In presence of this rebellious case of auricular catarrh, and after having minutely examined the buccal and nasal passage, I decided to try surgical intervention, believing that there was a purulent collection, of unknown origin, in the ear. The dog was anæsthetized by an intra-peritoneal injection of a solution of chloral and morphine, and an incision about 4 cm. long made at the level of the base of the ear and its external side. Nothing abnormal in the external ear; the membrane of the tympanum was punctured, a probe of small calibre introduced by this opening, and a brown awn extracted. By means of forceps three other brown hairs were extracted from the middle ear. The operation was concluded by irrigating well with oxygenated water and providing for drainage with some aseptic gauze. The lips of the wound were sutured, and a bandage put on, which was renewed forty-eight hours afterwards.

Unfortunately the dog died of pulmonary congestion some days later.

*Reflections on Anæsthesia of the Dog by Richet's Procedure.*

The method employed to anæsthetize the animal consisted in an intra-peritoneal injection of chloral and morphine by means of

a Pravaz syringe, and with all antiseptic precautions. In this procedure the doses were: Chlorhydrate of morphine, 2·5 milligrammes per kilo of the animal; chloral, 5 decigrammes. Three to four minutes after injection complete anæsthesia was obtained, immediately followed by syncope, causing an apparent state of death: complete arrest of respiratory movements, suppression of the corneal reflex, pulsations of the femoral artery very rare and hardly perceptible. Artificial respiration practised immediately; rhythmic tractions of the tongue, extension and reflexion of the anterior members, and moderate pressure on the thorax. This manœuvre carried on for two hours and a quarter. At this time respiratory movements reappeared slowly, as well as the corneal reflex.

Reflecting on this anæsthetic accident, probably due to too large a dose of chloral, the following formula is advised: Chlorhydrate of morphine, ·5 grm.; chloral, 10 grm.; water, 100 grm.

Inject 1 c.c. of this solution per kilogramme of the animal weight into the peritoneum. This solution has always given excellent results, and never occasioned accidents.

This observation has appeared interesting to us from three points of view:—

(1) The fact of having encountered in the middle ear a foreign body made up of brown awns.

(2) Practise anæsthesia of the dog cautiously with the solution of chloral and morphine.

(3) When one encounters a rebellious auricular catarrh in a young dog, one should remember the possibility of a foreign body in the ear, and not hesitate to intervene surgically.

(*Revue Générale de Médecine Vétérinaire.*)

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## Books and Periodicals, &c., Received.

Journal of the Royal Army Medical Corps; Transvaal Agricultural Journal; Agricultural Journal of the Cape of Good Hope; L'Étoile Belgique; Bulletins of the Bureau of Sleeping Sickness; Bureau of Animal Industry (U.S.A.); Board of Agriculture and Fisheries; Department of Agriculture and Technical Instruction for Ireland.

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## Letters and Communications, &c.

Mr. E. Bell, Anti-Bearing Rein Association; Dr. Burton Rogers, Kansas Veterinary Medical Association; Dr. Guisseppe De Cortes (Sardinia); Mr. S. Stockman; Mr. W. Cargill Patrick; Mr. W. Moodie; Dr. Tims; Professor Gofton; Professor O'Connor; Mr. N. Meyers; Dr. Stapley; Mr. J. Perry; Captain Leaning; Captain Swanston.

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